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FAVUS IN MICE AND MEN.

By J. IVAN CONNOR, M.B., M.S. (Melbourne), D.T.M. & H. (Cantab.).

(From the Walter and Eliza Hall Institute, Melbourne.)

RINGWORM in men handling wheat during the recent mouse plague in wheat-growing areas of Victoria and New South Wales has been prevalent where mice were numerous. A certain proportion of the mice have been found affected with skin lesions. Herman Lawrence[1] of Melbourne and Paul[2] of Sydney investigated a similar epidemic in 1918. Priestley[3] states that favus has been reported on two occasions in human beings in Australia, and by Cleland in mice prior to 1917. Dr. Lawrence made a very complete investigation, isolated the infecting fungus from mice and men in pure culture, reproduced the disease in both hosts and described the cultural appearance of the infecting fungus on gelatine. He concluded that the disease was favus due to infection with *Achorion quinckeanum*. Dr. Paul isolated a previously undescribed fungus which he named *Trichophyton rodens*.

An investigation during the last four months has revealed some further information. A visit in July last to the wheat stacks at Williamstown, Melbourne, where much of the wheat for shipment overseas has been stored, and a search for infected mice showed that though mice were numerous, very few had any skin lesions. (An afternoon's search produced two, and later by courtesy of Dr. Coutts, of Williamstown, six more infected mice were obtained.)

Some of the men at work rebagging the wheat had circinate ringworm-like lesions on neck and forearms. The lesions in the mice consisted of dry yellow crusts about the nose and ears, all more or less circular in outline, which on removal left a moist oozing surface. Under the high power of the microscope the crusts were seen to consist of a felted mass of spores and mycelium of a fungus.

Various bacteria and two different fungi grew in culture on Sabouraud's maltose agar medium: (i) A woolly rapidly growing fungus which later produced black terminal ascospores. (ii) A slow growing snow-white fungus.

The first of these was non-pathogenic to mice and guinea-pigs, but the latter when inoculated into mice, produced lesions similar to those found in the wild mice, that is, yellow crusts about the nose, eyes, ears and also on the legs. (Figure I.) These lesions were typical scutula of favus. The first two successfully inoculated mice were in the same cage, one died after three weeks and was promptly eaten by his

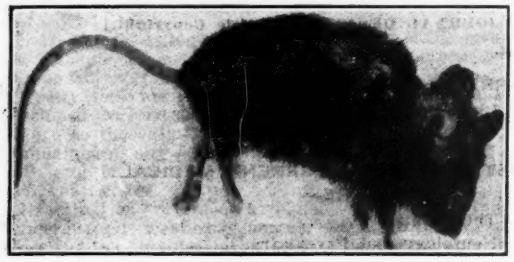

FIGURE I.
Laboratory mouse infected with *Achorion quinckeanum*, showing scutula.

companion, which lived for a further three weeks and then died of the infection. This appears to be typical of the course of the infection in the wild mice. Culture from the scutula of these mice on Sabouraud's media produced the same snow-white fungus. (Figures II and III.)

During the same period, by the courtesy of Dr. Wettenhall and Dr. Kelly, I was able to examine all the patients with ringworm in the outpatient dermatological clinics at the Melbourne Hospital. One patient had typical scutula in a circinate erythematous lesion on the forearm. Two other ringworm-like lesions were present without scutula on the same forearm.

Cultures on Sabouraud's media grew a snow-white fungus similar to that obtained from the mice. (Figure III, A.) This man had been pulling down an old sleep-out made of wheat sacks. Some of the other patients with ringworm gave a history of contact with wheat bags, but although fragments of mycelium were found in skin scrapings treated with potash 10%, cultures were sterile or grew other fungi of animal origin. I have not seen in my cultures anything resembling Dr. Paul's *Trichophyton rodens*.

FIGURE II.
Cultures of *Achorion quinckeanum* from laboratory mouse on maltose agar.

Cultures from a patient of Dr. Wettenhall showing typical scutula were handed to me by my colleague Miss Williams, of the Walter and Eliza Hall Institute. Those on subculture showed the same snow-white growth. (Figure III, B.) This man had been working on the wheat stacks at Williamstown in 1931 and then became infected.

FIGURE III.
Comparison of three cultures of *Achorion quinckeanum*. A = from man, B = from man, C = from mouse.

Microscopic examination of the cultures from mice and men with typical scutula of favus are all similar, showing septate mycelium and spores. (Figure IV.) The "favic tarsus" mentioned by Dr. Herman Lawrence may be seen in preparations from scutula, but not in culture. The fungus liquifies gelatine in forty-eight hours and grows fairly rapidly at 30° C. In hanging drop cultures the regular septate mycelium with terminal and lateral spores and sometimes a central endospore is seen (pectinate hyphæ and spirals are never observed). The cultures show a snow-white raised dome-shaped growth with a fluffy surface, which later develops a central depressed area. (Figures II and III.) The margin consists of a delicate fringe of growing mycelium. (Figure IV.) Photographs of achorion in Muir and Ritchie's "Manual of Bacteriology", 1927, grown from a cat and from man infected from the cat present appearances very closely similar to those obtained in the present investigation.

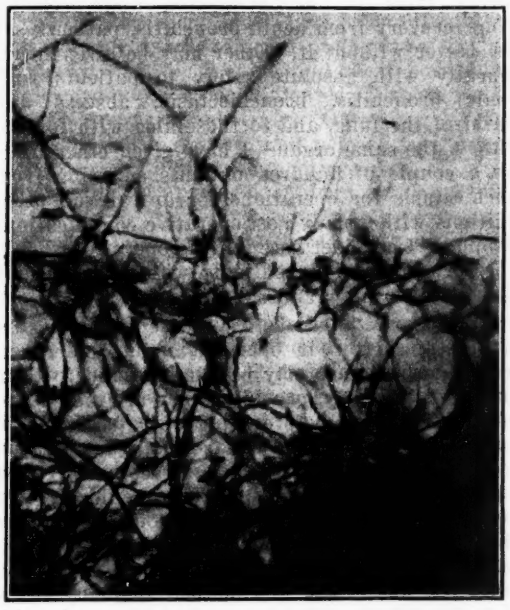

FIGURE IV.
Microphotograph of fungus *Achorion quinckeanum* from a culture on gelatine stained with borax methylene blue.

Ringworm in men working at the wheat stacks consists of circinate lesions usually on forearm and neck, the most likely sites of infection from abrasion and friction, though the hairy parts like the beard and scalp may also be infected. The general absence of typical scutula on the glabrous skin may be explained by the fact that very few lesions are left untreated. In the cases I saw at Williamstown pure lysol and iodine had been applied by the men themselves.

Bodin, Fox, Blaxal and Sabouraud,[3] cited by McLeod, have described circinate lesions on the skin and lesions like kerion on the scalp similar to those produced by the *trichophyta*, which on cultivation gave a culture like that of favus. Adamson, cited by Sequeira,[4] states that most of these cases of favus of the glabrous skin are not due to *Achorion schonleinii* but to the mouse achorion, *Achorion quinckeanum*. Sequeira[4] also states that accumulated stores of wheat infested with mice in Australian ports at the end of the war was the focus of infection by favus of many children paid to kill the rodents, but quotes no authority for the statement.

Diagnosis of Disease in Man.

The lesions caused by these infections vary considerably in appearance—yellow crusts or scutula may be seen in a small proportion of cases, more commonly circinate erythematous scaly lesions are present on the glabrous skin and kerion-like lesions on the hairy parts. The diagnosis of ringworm may be confirmed in the early stages of their evolution by microscopic examination of scrapings from the margin of the infected areas, placed in 10% potash.

Conclusions.

The probability is that mice are prevalent in the wheat-growing areas in Australia during the entire year. In good seasons they multiply and become a veritable plague. The parasite of favus, *Achorion quinckeanum*, has been present among wild mice at least since 1917,[1] and since then has spread in epidemic waves as they multiply and die off. Only a small proportion of the men exposed to infection contract the disease, which may be due to individual susceptibility. Typical scutula appear in a certain proportion of the lesions if untreated.

Treatment.

An ointment containing benzoic acid 1·0 gramme (15 grains), salicylic acid 1·0 gramme in vaseline 30 grammes (one ounce) is efficient for lesions on the glabrous skin. Kerion of hairy parts is better treated with mild antiseptic dressings of eusol or weak solution of perchloride of mercury, as the acute inflammatory reaction in these areas destroys the fungus.

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ANÆSTHESIA FROM THE GENERAL PRACTITIONER'S POINT OF VIEW.

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I HAVE written this paper, not for the use of the expert, but for the benefit of the general practitioner.

Within the last two or three years the world has been flooded with many new anæsthetics, and it is hard for the ordinary practitioner to get an unbiased opinion as regards their use. Every maker

extols his goods, saying they are the best in the world. Furthermore, if they are not patent drugs, the maker of gas anæsthetics strongly advocates the use of the gaseous anæsthetics, and often speaks disparagingly of the older, well tried out drugs.

Here again these people are influenced purely by commercial reasons. There is very little profit in the sale of chloroform, ether and ethyl chloride, as these are now more or less reduced to cut prices, with very little profit to the manufacturer, while the gas anæsthetics return a much bigger percentage of profit on a larger outlay of capital. For example, ether anæsthesia lasting an hour will probably cost one-tenth of what nitrous oxide anæsthesia lasting an hour would cost. This is the real reason why the various makers of drugs and anæsthetics are flooding the profession with their literature.

I do not speak disparagingly of nitrous oxide and ethylene, but they are not the drugs to be used by the general practitioner, unless he has been trained in their use and has given a large quantity of anæsthetics and has a big enough anæsthetic practice to warrant the outlay. Should his practice warrant the expenditure, and should he be able to command fees sufficiently large to pay for these higher priced anæsthetics, he will undoubtedly find them useful addenda.

To my mind, ethylene has been the biggest advance in anæsthetics in recent years, but there are certain things which will eliminate its use in general practice: (i) danger of explosion, (ii) the weight of anæsthetic machines to be carried about, (iii) lack of skill by the man who is not using it every day, (iv) the cost of apparatus and gases.

Ether has been a well tried servant and it has very seldom let us down. It may not be nice to take, but, if preceded by ethyl chloride, it is not at all unpleasant. If glucose and orange juice be given an hour or two before the operation, post-operative vomiting will be reduced to a minimum, especially if the ether is being administered by means of a machine.

The danger of post-operative pneumonia in this country is very small indeed, providing the patient is kept warm before, during and after the operation, and also that the temperature of the theatre is kept up to 21·1° C. (70° F.). I consider this most important. For a number of years post-operative pneumonia occurred at the Royal Prince Alfred Hospital in about three to five per ten thousand cases—less than the ordinary incidence of life. This was when ether was for the most part solely used as a routine.

With regard to preliminary medication with morphine, I have come to the conclusion that patients are better without it, for, if anything goes wrong, there is only one toxin for them to deal with. For example, ether can be readily eliminated from the patient's system, particularly if it is given by the intratracheal method, as it is a volatile anæsthetic. Morphine takes a long time to be eliminated and depresses the respiratory centre; and I have regretted in a few cases having previously administered morphine, as I have had a very uncomfortable time for a half to one hour. This is purely a matter of opinion, and numbers of people give morphine without any trouble; but I think that sooner or later they will come to the same conclusion as I have.

When the newer drugs were introduced into the Royal Prince Alfred Hospital the mortality rose from three per ten thousand cases to twelve to fifteen per ten thousand cases. This was alarming and more care and discrimination were taken in prescribing them.

There are certain myths about ether, started often by people who are physicians, but who know nothing whatever about anæsthetics. One of the myths is that ether should never be given to patients with lung disease. This is probably true of the acute conditions, for example, acute tuberculous disease. Here it is undoubtedly bad. During recovery from bronchopneumonia, and probably during recovery from acute bronchitis, patients are much better without it. Ether may be given intratracheally with absolute safety to patients with chronic bronchitis, bronchiectasis, abscess and hydatid of the lung, and to those also with foreign bodies in the same organ. I have given ether to at least a couple of hundred patients suffering from bronchiectasis for operations for antral and other conditions without any post-operative complications or trouble. I consider here that no operation should be performed on these patients which will occupy longer than an hour and a half.

With regard to diabetics, these patients are undoubtedly better if operated upon under gas anæsthesia, but ether may be administered to them with complete safety, providing they are dieted and treated a few days before being operated upon, so that their urine will be sugar-free a day or two before the operation. Two or three hours before the anæsthetic is administered they should be given fifty grammes of glucose by the mouth or rectum and twenty-five units of insulin by injection. If this is done, there is no danger of any post-operative trouble. I have used this method in many cases, and it is reliable. Be sure the patient takes his glucose, as I have known patients, not liking the sweetness of it, to throw away the glucose surreptitiously and so to suffer from hypoglycæmia.

"Avertin."

Concerning the newer drugs, there is one over which there seems to be a considerable amount of dispute, namely, "Avertin". The public, in certain cases, are clamouring for it. Some practitioners are enthusiastic about it, while others speak disparagingly about it. In this case, what is the practitioner to do?

In my opinion "Avertin" is a very useful adjunct to our armamentarium, but it must not be given in full anæsthetic doses. It is sold as a basal anæsthetic and must be used as such. Always have a good working margin of ether anæsthesia which you

can control, and on no account give any other drugs, such as morphine, with it. The dose should be 0·08 and occasionally 0·1 gramme per kilogram. If this small dose is used, you will have a safe working margin for ether anæsthesia, followed by a period of post-operative analgesia and sleep of about three to five hours, during which there is generally very little vomiting.

If patients with acute lung conditions have to be operated upon, "Avertin" may be given in slightly larger doses and a little less ether may be used, when it will be found that no harm will result.

"Avertin" has one disadvantage, that the patient requires skilled attention for about five hours after he has left the theatre. This is a drawback to its use in under-staffed and small hospitals.

I feel that a good deal of the trouble that has occurred with "Avertin" has been due to lack of care and precision.

When the patient is weighed, he should be weighed naked, and the scales should be reliable. Secondly, the drug must be accurately measured out, and thirdly, the temperature must be accurately estimated. If this is done, and the "good enough", haphazard method is eliminated, I think that the drug is comparatively safe. One toxin is enough in the patient's system, and neither morphine nor any other preliminary hypnotic should be given.

"Avertin" is an excellent drug for exophthalmic goître cases, but, being a respiratory depressant, it is safer to follow it by intratracheally administered ether.

"Sodium Amytal."

With regard to "Sodium amytal", this drug is also very useful, if not given in too big doses. The rule I make is to give 0·18 gramme (three grains) by the mouth the night before operation, followed by 0·18 gramme a couple of hours before the operation, or to very big men, 0·36 gramme (six grains). These patients can generally walk into the operating theatre. They have very little fear and feel well.

"Sodium amytal" being a barbitone preparation, its administration is often followed by forgetfulness, so that many patients do not remember very much about going to the operating theatre. Another advantage is that they sleep well after the operation and have plenty of time to eliminate most of the ether before they regain consciousness, so that when they wake up they feel quite well. Again, what I said about morphine being administered with "Avertin" holds good with this drug.

When giving ether intratracheally after the administration of "Sodium amytal", it will be found that the larynx is much less sensitive, and it is generally quite easy to pass the intratracheal tube. The interpretation of the eye reflexes and pupils after the administration of "Sodium amytal" is a little difficult at first. The patients have often no corneal reflex when anæsthesia is comparatively light. If the pupil is half dilated, anæsthesia may be either too light or too deep. Therefore, great care must be exercised to determine the stage of anæsthesia.

Proper surgical anæsthesia is generally obtained when the pupil is small. Remember that "Sodium amytal" is a respiratory depressant, and, if possible, it is safer to use intratracheally administered ether in conjunction with it, or some method in which, say, 5% carbon dioxide can be administered with oxygen.

I have seen very serious trouble with "Amytal" given intravenously. Rules for the use of "Sodium amytal" are as follows:

1. Use it as a basal anæsthetic.
2. Do not use morphine with it.
3. Do not give too much.

If you follow these rules you will have no trouble.

Spinal Anæsthesia.

Coming next to spinal anæsthesia, this method has its uses, but should never be used as a routine method, because it is much more likely to be followed by pulmonary complications than inhalation anæsthesia. Furthermore, it always causes some pathological changes in the spinal cord and nerve roots.

The following is a summary of Brown and Debenham's work on pulmonary complications following inhalation and spinal anæsthesia.

1. In 812 cases post-operative pulmonary complications were 4·29 times more frequent after subarachnoid anæsthesia than after inhalation anæsthesia, in spite of the fact that more "bad risk" patients were operated on under inhalation anæsthesia.

2. The adverse ratio for subarachnoid anæsthesia is found, regardless of the region of the body operated on or the type of operation performed.

3. The more closely the operative procedure approaches the diaphragm, the greater is the incidence of post-operative pulmonary complications.

Regional Anæsthesia.

Lastly, we come to local and regional anæsthesia. This method is not entirely free from risk. In the last two years I have known of two sudden deaths occurring after injections of local anæsthetics. Whether the cause is due to the adrenaline or the anæsthetic, it is very hard to say. I have given a dog of forty-five pounds weight 0·49 gramme (three-quarters of a grain) of cocaine intravenously without causing sudden death. It is possible that the adrenaline entering a vein may be the cause.

Conclusions.

To sum things up, one may say that ether is a most excellent anæsthetic. It is safe, portable and cheap. It has stood the test of time and is being attacked largely by the commercial houses, not for any benefit to humanity, but for their own gain.

Ethylene is probably the safest of all anæsthetics, but is not suitable for general practice.

"Avertin" and "Sodium amytal" are excellent basal anæsthetics, but must be measured carefully and not in a haphazard manner, and should never be used, except under exceptional circumstances, for full narcotization.

LARYNGEAL DIPHTHERIA.

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I AM reporting shortly the experiences during four months' intake at the Royal Alexandra Hospital for Children in the ward set apart for patients with laryngeal diphtheria.

The condition is common at all times of the year. Two hundred and six patients were admitted to hospital during the four months from March to June of this year; fourteen of these died. The ages varied between nine months and ten years. Two patients were reported to have had diphtheria previously, and one child was admitted twice during this time with diphtheria. The duration of the illness before admission to hospital varied between one and ten days. The majority of patients had been ill three or four days before admission; and practically all of these were sent for admission without delay by the medical practitioner who saw them first.

The reason for a somewhat prolonged illness before admission, in many cases, was that the mode of onset varied greatly; the parents often did not suspect any serious disorder, and did not summon a medical practitioner in the early stages. I quote the symptoms in some typical cases:

S.F. Ill for three days with cough, croup, vomiting, loss of appetite, noise in the throat and difficult breathing.

E.K. Ill for four days, drowsy, vomiting, husky, with loss of voice and heavy breathing.

J.M. Ill for ten days with sore throat, vomiting, loss of voice, cough, loss of appetite, croupy cough and difficult breathing.

J.T. Ill for one day, croupy, sore throat, could not get his breath, retchings, loss of voice.

A.L. Headache for seven days, sore throat for three days, vomited today, has been listless and thirsty.

L.F. Bronchitis nine days ago, loss of voice for two days, distressed breathing for two days.

As a rule advice was not sought until the breathing became distressed; this occurred at any time from the first to the tenth day in this series.

Two children had ulcerative stomatitis for some days prior to the development of laryngeal symptoms.

The signs on admission were most commonly, laryngeal stridor, cough, husky voice or cry, loss of voice, fever up to 38·35° C. (101° F.) and 39·45° C. (103° F.), rapid pulse, and recession of the chest wall on inspiration. Membrane was present on one or both tonsils or on the pharynx in most cases, but no membrane was seen in a large number. Throat swabs were taken on admission to hospital and every twenty-four hours until diphtheria bacilli were found, or until three swabbings without diphtheria bacilli had been reported.

In several cases when adherent membrane was present on the tonsils all throat swabs gave negative results; in many others no diphtheria bacilli were found until the third day: therefore the diagnosis was always made on clinical grounds, based mainly on the laryngeal stridor, loss of voice or inability to cry, and recession. Any child presenting these signs was given 20,000 units of diphtheria antitoxin at once, and 10,000 units every twenty-four hours afterwards, until definite clinical improvement was noted. Rarely more than 50,000 units were given.

All these children with obvious recession on inspiration were placed in a cot inside a steam tent on admission to hospital; the steamy atmosphere was maintained continuously until improvement occurred or until it was apparent that no relief was being afforded. In these cases intubation was usually necessary, or failing intubation tracheotomy was performed.

Laryngeal diphtheria was to be differentiated principally from simple laryngitis or croup.

There was no certain method of differentiating simple laryngitis from diphtheric laryngitis, in the early stages. Observation revealed that as a rule simple laryngitis subsided more rapidly in a steamy atmosphere.

With a history of previous attacks of croup, with large tonsils, a flushed face, and no membrane, it was probably safe to observe the child for a few hours in a saturated steamy atmosphere.

As regards prognosis, marked pallor, extreme recession of the chest wall and a pulse rate over 160 per minute, especially if the pulse was irregular, were serious signs. The normal sinus arrhythmia of childhood was misleading in the acute stages, however. The temperature was rarely over 39·45° C. (103° F.) in diphtheria alone. The respiratory rate when recorded was about 40 per minute when the temperature was above 38·35° C. (101° F.). With a respiratory rate continuously over 50, pneumonia was suspected. Apart from pneumonia and a rapid or irregular pulse, complications were rare. In this series, one child had loss of power in all four limbs, especially in the legs, with foot-drop, which lasted three or four weeks; four children had a temporary squint; two had regurgitation of fluids through the nose; two children developed the rash of scarlet fever within thirty-six hours.

We were struck by the infrequency of complications.

The mode of onset reported by the parents was very variable—vomiting, diarrhœa, cough, sore mouth, running nose, husky voice, difficult breathing.

The lives of several of the patients might have been saved if the medical attendant had been called earlier in the disease.

It was noteworthy that the majority of the children were sent to hospital, with a tentative diagnosis of

TABLE I.
Fatal Cases.

Membrane.	Age in Years.	Day of Illness.	Intubated.	Tracheotomy.	Died on days after Admission.
?	2	7th	Yes.	Yes.	1
Yes.	10	9th	Yes.	Yes.	1
Nil.	8	2nd	Yes.	Pneumonia.	8
Nil.	3	3rd	Yes.	Yes.	2
Yes.	7	4th		Pulse fell to 70.	2
Yes.	4	3rd	Yes.	Pneumonia.	2
Yes.	9 months.	1st	Yes.	No.	2
Yes.	14 months.	7th	Yes.	Yes.	1
Yes.	14 months.	2nd	No.	No.	1
Yes.	3	2nd	No.	No.	1
Yes.	9	6th	No.	No.	1
Yes.	18 months.	7th	Yes.	No.	1
Yes.	9	4th		Pneumonia.	5
Yes.	7	22nd	No.	Pulmonary tuberculosis.	18

TABLE II.
Outstanding Symptoms in Consecutive Cases.

Age.	Units.	Duration.	Recession.	Throat Swab.	Membrane.
4 years.	20,000	Cold for two days, husky voice.	Recession.	Positive.	+
16 months.	20,000	Four days, croup.	Slight—5th day.	Negative.	−
22 months.	10,000	Stridor for one day.		Negative.	Nil. Croup.
8 years.	20,000	One day, cold, sore throat, voice lost.	−	Negative.	+
3 years and 5 months.	20,000	Three days.	+	Negative.	+
3 years and 2 months.	20,000	Two weeks, cough.	−	To Coast.	+
2 years.	20,000	Three days, fever, vomiting, hoarseness, dyspnœa.	−	Negative.	−
3 years.	20,000[1] 10,000 10,000 10,000	Three days, sore throat.	Stridor + +	Tubed; negative.	+
6 years and 6 months.	20,000[1] 10,000 10,000	Four days, drowsy, cold, vomiting, croupy.	−	Negative.	−
1 year and 8 months.	20,000	One day.	Tonsils large. No membrane.	Negative.	Nil. Croup.
3 years and 6 months.	20,000	One week.		Negative.	Nil. Croup.

[1] The several doses used in this case were given on successive days.

diphtheria, very soon after being seen by the medical practitioner for the first time.

In some cases, however, the indications of diphtheria seem to have been preceded by symptoms by no means suggestive of that condition.

Further education of the public towards obtaining medical advice early for children's ill-health is obviously necessary.

I have recorded some particulars of (i) fatal cases; (ii) outstanding symptoms in consecutive cases; (iii) typical histories.

Typical Histories.

S.F., aged three years, was admitted to hospital on March 13, 1932, and on March 21, 1932, was discharged and sent to the Coast Hospital. Examination revealed substernal and intercostal recession and audible laryngeal stridor. Diphtheria antitoxin was administered as follows: March 13, 20,000 units; March 14, 10,000 units; March 15, 10,000 units. The patient was placed in a steam tent from the time of admission. There was persistent vomiting for two days. Intubation was performed on March 15, giving great relief to the patient; the tube was removed after two days. On examination no membrane was found; a culture taken from a throat swab revealed the presence of diphtheria bacilli. The patient's temperature on successive evenings was 37·8° C., 39·45° C., 38·35° C., 37·8° C. and 37·25° C. (100° F., 103° F., 101° F., 100° F. and 99° F. respectively). The pulse rate on admission to hospital was 146 per minute, while 164 was the maximum rate recorded; it was 120 on the fourth day and 116 on the eighth day. The patient recovered.

E.K., aged eight years, was admitted to hospital on February 26, 1932, and was discharged and sent to the Coast Hospital on March 1. Examination revealed adherent membrane on both tonsils; there was recession, stridor and partial loss of voice. On February 26, 30,000 units of diphtheria antitoxin were administered; and on February 27, 10,000 units. No diphtheria bacilli were found in three successive throat swabbings. The pulse rate ranged from 140 to 150 per minute. The temperature was 38·35° C. (101° F.), reaching a maximum of 39·16° C. (102·5° F.).

J.M., aged six and a half years, had had a sore throat for ten days, together with vomiting, aphonia, cough, anorexia, croupy cough and difficult breathing. On February 22, 1932, 20,000 units of diphtheria antitoxin were administered; and on February 23, 10,000 units. Membrane was present. On the third day diphtheria bacilli were found in the throat swabbing. The temperature was 38·35° C. (101° F.) on two days. The pulse rate on admission to hospital was 140 per minute; on the third day it was 110; on the ninth day it was 110 and on the sixth day it was 90.

J.T., aged five years, had been croupy for one day, with a sore throat; he could not get his breath and there were retching and loss of voice. On the first day 20,000 units of diphtheria antitoxin were given. Examination revealed stridor, croupy cough and aphonia. On the third day diphtheria bacilli were found in the throat swabbing; there was no membrane. The patient's condition rapidly improved.

THE SIMULATION OF PITUITARY DISEASE BY INTRACRANIAL LESIONS.

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DIAGNOSTIC difficulties in respect to lesions in the region of the *sella turcica* are well known, but of recent years, owing to the increasing frequency of surgical exposure of this region and to the detailed study of numerous cases by neurosurgeons, particularly by Cushing and his school, a number of symptom complexes can now be recognized with the possibilities of great improvement in our diagnosis. By a careful correlation of the clinical aspects, the radiographic appearances, findings at operation, and the pathological picture, an extremely detailed knowledge of these tumours has been acquired, so that it has become possible, not only to localize them accurately, but also to make a definite statement as to their probable pathological nature in the majority of cases. It is thought, therefore, as much of the literature is not readily available, that a short review of the subject with some illustrative cases would be of interest to Australian clinicians generally.

Pituitary Lesions Proper.

Since the publication of Cushing's classical monograph on "The Pituitary Body and its Disorders",[1] the signs and symptoms of primary pituitary lesions have become well known. They consist of:

1. A glandular syndrome, which is due to interference with pituitary function, with the production of disturbances of growth and metabolism. Naturally these show great variation, dependent in the main on the age of the patient.

2. A chiasmal syndrome, which is due to various pressure effects on the optic nerves, the chiasm, or the optic tracts and which is manifested by progressive changes in the eye grounds and defects in the visual fields.

3. Focal or general pressure syndromes, which are due to intracranial extension of the tumour, with

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on June 30, 1932.

pressure on the infundibulum, the third ventricle, the frontal or temporal lobes, or on various cranial nerves.

It may be stated that in general the above is also the order of appearance and of relative importance of these main syndromes: in other words, general intracranial extension is a late development and may often be entirely absent.

It has, however, long been recognized that any one of these may be the initial syndrome, that any one may be absent, or that any one may predominate in the clinical picture. The following case history illustrates this:

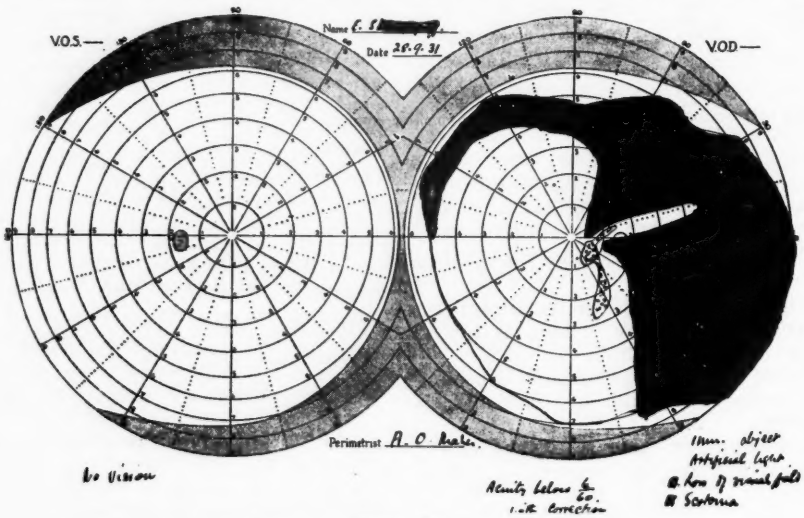

FIGURE I.
Showing fields of vision in Case I, September 20, 1931, before operation.

CASE I.—E.S., a male aged fifty-six, was admitted to Saint Vincent's Hospital under the care of Dr. Douglas Miller on September 28, 1931. He complained of loss of vision in the left eye, which had progressed to complete blindness during a period of two years. Six months before admission to hospital he had noticed impairment of vision of the right eye, which had so progressed that when first seen he was unable to read, although he could discern objects faintly. He also suffered from occasional frontal headaches. Otherwise he was in good health.

On examination he exhibited no signs of any endocrinal disturbance that could be attributed to dysfunction of the pituitary gland, although in view of his age it was difficult to exclude mild hypopituitarism. Examination of the eyes revealed bilateral primary optic atrophy, the atrophy of the left optic nerve being more advanced than that of the right. The visual acuity of the left eye was nil, of the right $^{6}/_{60}$, while examination of the fields of vision showed a temporal hemianopia in the right eye with a central scotoma. (See Figure I.) There were no mental changes, no signs or symptoms referable to the other cranial nerves, and no signs of increase of intracranial pressure.

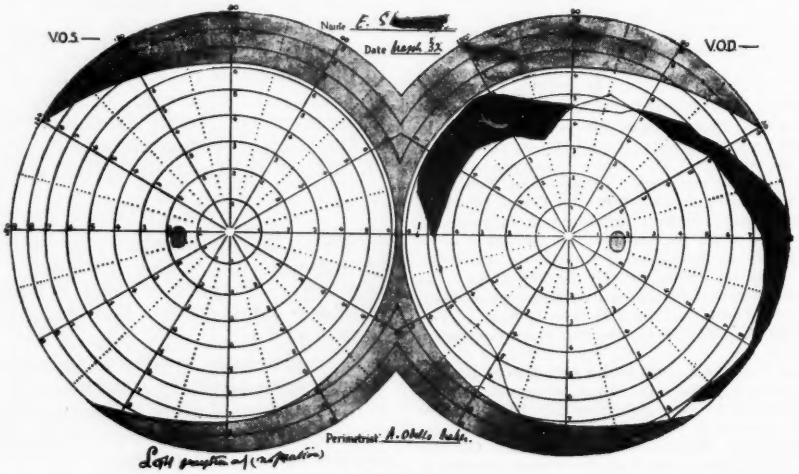

FIGURE II.
Showing fields of vision, Case I, four months after operation.

Radiography revealed general enlargement of the *sella turcica*, with little if any alteration of the contour of the clinoid processes.

The diagnosis of chromophobe adenoma of the pituitary was confidently made, and on October 13, 1931 a transsphenoidal removal of part of the adenoma with consequent decompression of the gland was carried out by D.M. and H.R.D. The immediate result of the operation was excellent, the patient rapidly regaining very good vision in the right eye and some vision in the left so that even with this eye he was able to recognize objects readily. (See Figure II.) When he was seen four months later,

ILLUSTRATIONS TO THE ARTICLE BY DR. HAROLD R. DEW.

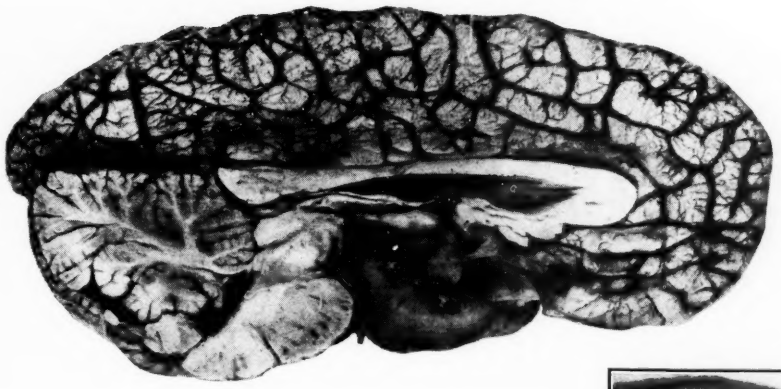

FIGURE III.

Mesial section of the brain, Case II, showing site and size of the suprasellar tumour.

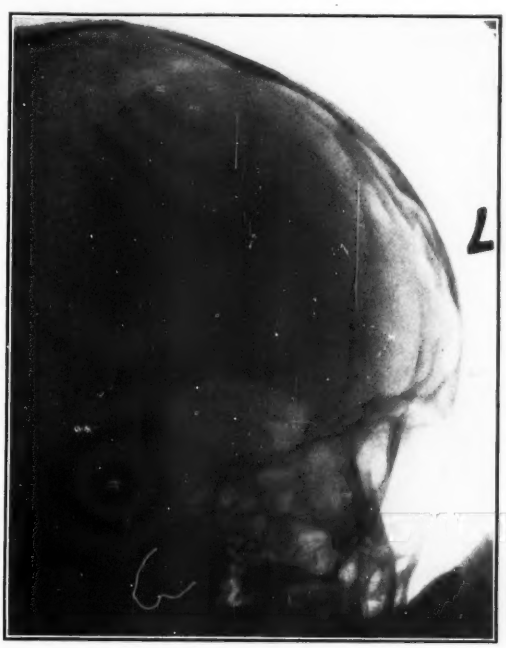

FIGURE IV.

Radiograph in Case III, showing great enlargement of the left optic foramen. (By courtesy of Dr. H. Sear.)

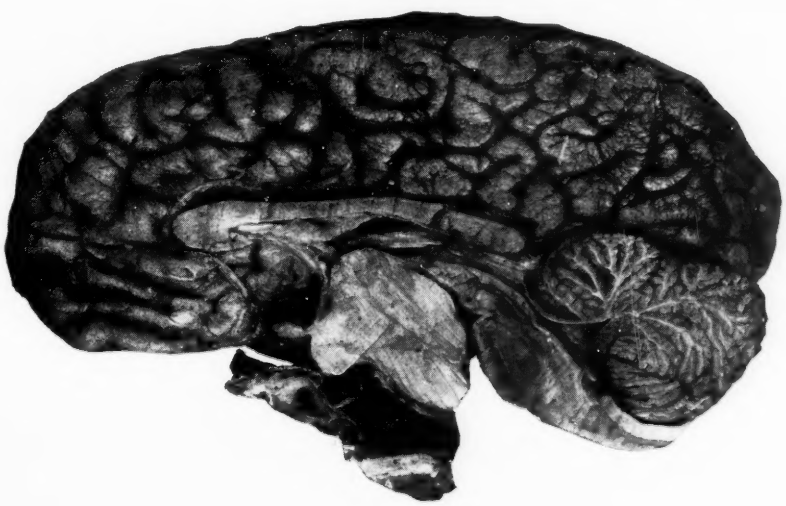

FIGURE VI.

Mesial section of brain, Case IV, showing the relations of the third ventricle tumour.

his improvement was maintained. The pathological diagnosis was chromophobe adenoma.

Such a case is typical and representative of a group in which there should be little doubt either as to the correct diagnosis or the method of treatment. It may be otherwise, however, in the case of those growths which, lying in a suprasellar position, may produce a variety of clinical syndromes.

SUPRASELLAR TUMOURS.

There are a number of different tumours which, owing to their site or origin, may produce syndromes similar to those referred to above, and so closely simulate true pituitary disease. In these, however, the order of appearance and the relative importance of the syndromes are different: in the majority of cases the chiasmal syndrome is the first recognized. In spite, too, of the varied effects produced, it is usually possible by clinical methods to differentiate all these from each other and from primary pituitary disease, particularly on the evidence of age incidence, clinical history, physical signs and X ray appearances. It is essential to attempt to diagnose not only the site, but also the pathological nature of the lesion, as it is extremely important for the surgeon to have some idea of the type of lesion he may expect to find. In all of these tumours the chiasmal syndrome is the most important, being associated nearly always with an essentially normal sella.

Before considering them in detail, it is advisable to refer to some important anatomical studies that have been recently carried out, as these serve to explain some of the variability of the symptomatology of chiasmal pressure. These are first the observations of de Schweinitz[2] and Schaeffer,[3] who have shown that the optic chiasm exhibits some anatomical variations.

With the chiasm in the common position, a tumour originating in the sella presses upwards, involves the decussating fibres and ultimately causes a more or less symmetrical bitemporal hemianopia. In the post-fixed type either or both optic nerves may be involved in front of the chiasm, with as a rule progressive blindness in one eye and temporal hemianopia in its fellow. Less commonly the chiasm is prefixed, although in spite of many statements in anatomical text books it never, except as the result of pressure posteriorly, comes actually to occupy the chiasmatic sulcus. In these cases a tumour may present behind the chiasm, so that either it or the optic tract may be pressed upon, in the latter case with the production of an homonymous hemianopia. Studies of the arrangement of the bundles in the chiasm have also been made by Traquair[4] and others, and it is now conceivable that the position of any suprasellar growth, as related to the chiasm, may come to be determined solely by the character of the field defects, for if, as it is claimed the crossed macular bundles lie in the posterior part of the chiasm, a macular scotoma may be more likely to precede the loss of vision in the outer temporal field in a posteriorly placed lesion than in one placed anteriorly. Another point of great importance is the variability of the distance between the chiasm and the sellar diaphragm. This varies from actual contact to a distance of ten millimetres with an average of five millimetres, so that the pituitary gland in some cases has to increase to double or treble its size before it exerts any pressure on the chiasm.

TYPES OF LESIONS SIMULATING PITUITARY DISEASE.

The types of lesions simulating pituitary disease may be summarized as follows:

A. Suprasellar tumours.
  1. Tumours of the hypophyseal duct or cranio-pharyngiomata.
  2. Suprasellar meningiomata.
  3. Gliomata of the optic chiasm.

B. Adjacent lesions.
  1. Tumours involving the anterior part of the third ventricle or infundibulum.
  2. Meningiomata of the olfactory groove.

C. Distant lesions.
  Intracranial tumours producing internal hydrocephalus.

D. Rarer lesions—non-neoplastic in nature.
  1. Aneurysm of the arteries of the circle of Willis.
  2. Chronic basal cisternal arachnoiditis.

Hypophyseal Duct Tumours.

Hypophyseal duct tumours are essentially epithelial tumours derived from remnants of the hypophyseal or craniopharyngeal duct, and are often called tumours of Rathke's pouch or cranio-pharyngiomata. The development of this region has been fully reviewed by Erdheim,[5] who emphasizes the fact that owing to the rotation of the developing gland, the remnants of the hypophyseal duct tend to become displaced cranially and ventrally until they come to lie ventral to the infundibulum. As pointed out by Duffy[6] and Critchley and Ironside,[7] the two chief sites for remnants of this structure are in this situation.

There seems little doubt that the entire region of the hypophysis from the pharynx to the infundibulum is peculiarly rich in vestigial epithelial *débris* which may be considered to have the potentialities of glandular function and of neoplastic formation. Many cases of these interesting congenital tumours have been described and the reader is referred to the abovementioned articles of Duffy and Critchley and Ironside for an exhaustive discussion of them in all their aspects. Pathologically they show great histological variability: they may be solid or cystic, degenerative changes are not uncommon, while they may contain cholesterin or calcareous nodules, so that on cutting the tumour a gritty sensation is often imparted to the knife. As a rule they are truly suprasellar, giving rise as a result to pressure effects on the chiasm and the anterior part of the third ventricle.

Most commonly the upper epithelial *Anlage* is the starting point, and as the tumour grows the anterior wall of the third ventricle is pushed inwards, or the ventricle may be actually invaded by the growth. The pituitary gland is completely separated from the tumour and usually, apart from slight pressure flattening, appears normal. The chiasm lies at first in front of and later below the tumour, so that pressure on it is usually downwards and forwards.

The posterior clinoid processes gradually become absorbed and the sella may be converted into a shallow open bowl. In the rare cases in which the growth originates from the lower *Anlage* within the

sella, the effects and appearances are indistinguishable from that of a primary pituitary lesion. As is to be expected from its embryonal origin the histological picture may be complicated, and Duffy has described three distinct cellular types: (i) Benign squamous epithelial cysts, (ii) adamantinomata, (iii) spindle-celled carcinoma with malignant characteristics.

In most of these the epithelial nature of the tumour is never in doubt, although, as pointed out by Critchley and Ironside, the bulk may be made up of glial reactionary tissue with only here and there islets of epithelial cells which may, therefore, be difficult to find.

The resemblance of some of the more solid adamantinomata to similar tumours derived from epithelial remnants in the jaws is very striking.

Clinical Aspects.

Age.—As would be expected from their origin from embryonic rudiments, these tumours nearly always appear comparatively early in life, so that the age of incidence lies between ten and thirty years of age. Sometimes, however, growth appears to be very slow and a few cases have been recorded in patients of advanced years.

Initial Symptoms.—Visual defects are nearly always manifested by the appearance of a chiasmal syndrome due, in the case of the commoner truly suprasellar type, to pressure downwards and forwards on the posterior part of the chiasm. As a result the lower temporal quadrants of the visual field are probably first affected with or without a paracentral scotoma followed by a bitemporal hemianopia. The development of the visual defects in these and allied chiasmal lesions has been very accurately recorded and correlated by Cushing and Walker,[8] who have emphasized how commonly one eye is affected before the other. Very often the patient first presents himself with blindness in one eye and with temporal hemianopia in its fellow. In those growths which originate from beneath the sellar diaphragm the upward pressure on the chiasm would cause an exact simulation of primary pituitary lesions with involvement of the upper temporal quadrants first.

In marked contrast to intrasellar growths, in which primary optic atrophy in the eye first affected is the rule, these duct tumours, particularly in the younger patients, often give rise to papillœdema. It would seem that this is due to the relatively early production of pressure on the third ventricle, with resulting increase of intracranial pressure before the optic sheaths are completely shut off. In some adult patients, however, in whom no doubt very gradual pressure on the chiasm has been present for a long time without any involvement of the third ventricle, primary optic atrophy may arise without papillœdema, while in other cases a secondary papillœdema may be superimposed on a primary optic atrophy as a result of late great increase of intracranial pressure.

General Pressure Phenomena.—Owing to extension back into the interpeduncular fossa, compression of the third or sixth nerves, with resultant ocular palsy, pressure on the fifth nerve with facial neuralgia, or even pressure on the cerebral peduncles with various reflex phenomena can all occur. As has been noted, these effects may also all occur in the very late stages of primary pituitary growths.

In the same way, pressure on the anterior part of the third ventricle leads to involvement of the infundibular nuclei with the production in some cases of a hypothalamic syndrome; later, internal hydrocephalus with increase of general intracranial pressure takes place. It is obvious then that signs of general intracranial extension are much earlier in their incidence and are seen much more frequently in these tumours than in purely intrasellar growths.

Pituitary Symptoms.—In the case of tumours developing from the inferior *Anlage*, development of the anterior lobe of the pituitary may be interfered with, with the production of varying degrees of hypopituitarism. Compression by a truly suprasellar growth, however, may also lead to pituitary deficiency, particularly in the later stages. Owing to the age incidence of these tumours, this usually appears before puberty, manifesting itself as a mild degree of sexual and skeletal infantilism. The patients, however, show no mental backwardness. If the lesion occurs in adults there may be signs of genital dystrophy, obesity, and lowered basal metabolism. It is difficult to assess what symptoms may be induced by direct pressure on, or distension of, the infundibular region. Controversy still rages as to the exact relationship, but there is no doubt that a close functional connexion exists between the hypothalamic nuclei and the pituitary. This question will be discussed again later. It seems probable that the extreme drowsiness so commonly noted, the polyuria, the polydipsia and possibly the *diabetes insipidus* shown by some of these patients should be really regarded as hypothalamic in origin.

Toxic Symptoms.—In these duct tumours peculiar toxic effects are common: it has been tentatively suggested that these are due to the absorption of break-down products of the tumour. They take the form of irregular pyrexia, headache, urticarial rashes, and many vague syndromes which may simulate influenza, meningitis *et cetera*. It is a well known fact and one which is very disturbing to all neurosurgeons, that any operative interference with these growths, even a simple decompression, is often followed by uncontrollable hyperthermia, restless delirium, convulsions and death.

Radiographic Appearances.—Except in those tumours which have developed within the sella, in which the appearances are those of tumour of the pituitary, the sella may show little departure from the normal. It is true that in advanced cases there may be some erosion of the posterior clinoid processes, while the sella tends to be comparatively shallow, with a wider inlet and sometimes a little irregularity in the floor. What is of great importance, however, is the appearance in stereoscopic radiographs in at least 80% of these tumours of areas of calcification which show well above the sella.

Although the following tumour cannot be classed histologically as a cranio-pharyngioma, yet, both as regards its site and its clinical history it presents such an exact stimulation of these growths, that it is included here.

CASE II.—J.E., a male, aged seventeen years, was admitted to the Sydney Hospital under the care of Dr. G. Bell with a history of failing vision during the previous two years. The left eye was first affected and when the patient was seen at another hospital a little after the onset, the ophthalmological diagnosis was optic neuritis. He was seen by a rhinologist and his tonsils and adenoids were removed, there being well marked hæmorrhage at the time of operation. At about the same time antrostomy was carried out and for a while he believed he could see better out of the edges of the left eye. Shortly after this he was seen again by the same ophthalmologist at another hospital, when the discovery of double optic neuritis with papillœdema and a gross temporal field defect in the right eye led to the suggestion that he had an intracranial lesion and a request that he report for perimetric examination. He failed to do this, and since then the vision of the other eye had gradually failed. A year before admission to hospital an attack of vomiting occurred, which lasted for a period of some days, but there had been no vomiting for six months previous to admission. Occipital headaches had been severe for the last two months and for the last year he had been nervous and easily upset. There had been no loss of weight, no polyuria, and no symptoms directly referable to the organs of internal secretion.

On examination he was well nourished, possibly somewhat of a female habitus, the pubic hair was of the female distribution, but there was no genital dystrophy. The voice was apparently normal for his age. His visual acuity was very poor, so that perimetry was impossible, while both eyes showed advanced papillœdema. Nothing abnormal was detected in the other cranial nerves, while the only abnormal reflex phenomenon noted was an absent right superficial abdominal reflex. He gave the appearance of a low degree of hydrocephalus, his headache kept him very quiet, while, although there was no mental deficiency, he seemed dull and apathetic. There was, however, no uncontrollable drowsiness.

Radiographic examination revealed some convolutional atrophy of the inner table of the skull, slight atrophy of the posterior clinoid processes, but otherwise an essentially normal sella. No definite suprasellar calcified areas could be seen, although stereoscopic films were not examined. After a few days in hospital increased tone in the upper limbs was noted and a bilateral extensor plantar reflex developed.

The picture then was a clear-cut one. The age of the patient, the chiasmal syndrome, the development of hydrocephalus, the papillœdema, the destruction of the posterior clinoid processes with an essentially normal sella, led to the diagnosis of suprasellar tumour, probably derived from the hypophyseal duct.

The need for quick action to try to save the sight being recognized, and also the difficulty and danger attending on a direct attack on an advanced tumour of this nature, a palliative right subtemporal decompression was advised and was carried out by Dr. G. Bell under local anæsthesia. The patient, however, developed hyperthermia and restlessness, followed by death in coma forty-eight hours after operation. Autopsy revealed a large, firm tumour projecting from the infundibular region on to the *dorsum sellæ*, pressing from behind on the chiasm which was more or less incorporated in the tumour. The pituitary was not affected, either as regards size or structure, and was separated from the tumour by the sellar diaphragm. There was well marked secondary hydrocephalus, and the tumour had apparently originated from the region of the infundibulum. (See Figure III.)

Many sections of this tumour were cut and stained in a variety of ways. Instead of discovering some epithelial masses as I expected, we found the whole tumour to be a glioma. Although it gave the impression of being tough and fibrous in places, there is no doubt that the tumour is in the main comprised of bipolar spongioblasts, that is, it is a spongioblastoma. Although it has been recognized that some of the cranio-pharyngiomata may be composed almost entirely of glial reactionary tissue, yet in all such cases some epithelial structures are found. It is, therefore, certain that this tumour must be regarded as a primary glioma of the infundibular region, and not as a tumour derived from the hypophyseal duct.

Gliomata of the Optic Nerves et cetera.

Cushing and Martin[9] have recorded an interesting series of gliomata arising from the optic chiasm and adjacent wall of the third ventricle and have stated that these tumours comprise 8% of all suprasellar tumours which give a chiasmal syndrome. Verhoeff[10] has shown that many intraneural tumours of the optic nerve which have been previously recorded in the literature as neurofibroma, myxogliomata, myxoma, or myxosarcoma, should in reality be classed as gliomata, and most authorities now agree with him. Gliomata of various types can of course arise from the region of the anterior perforated space, the wall and floor of the third ventricle or the infundibulum, but according to Cushing these rarely actually invade the chiasm, and as they produce internal hydrocephalus relatively early, are associated with papillœdema rather than primary optic atrophy. The tumours under discussion tend to invade the nerve head and may produce a pear-shaped protrusion into the orbital cavity with erosion and enlargement of the optic foramen. In Cushing's series it was also noted that a certain number of the patients exhibited the cutaneous type of neurofibromata described by von Recklinghausen.

Clinical Aspects.

These tumours are almost entirely restricted to young children between the ages of four and fourteen, but owing to their slow growth often show a long survival period.

Visual Defects.—The first symptom is failing vision in one eye, and this is not infrequently associated with homolateral exophthalmos.

On examination primary optic atrophy is found in the affected eye; sometimes, owing to actual invasion of the nerve by the neoplastic process, an elevation of the disk can be detected. The vision of the other eye then becomes affected, going through the same atrophic changes in the disk, with sometimes, however, a variable period during which rather bizarre types of hemianopia may be present. Rarely apparently is a hemianopia with a vertical meridian discovered.

General Pressure Effects.—General pressure effects are due to gradual extension of the neoplasm into the interpeduncular space with the production of internal hydrocephalus, sometimes associated with an hypothalamic syndrome. Of course, ultimately there is evidence of pressure on one or other cerebral peduncle. Pituitary glandular symptoms are usually absent and, if they are present, they are simply those of mild compression deficiency with a low grade hypopituitarism. The condition is usually slowly progressive and for long the main symptom may be blindness only.

Radiographic Appearances.—As a rule radiographic examination reveals an essentially normal, unexpanded sella, although in some cases there may be a pear-shaped extension of the cavity forward under the anterior clinoid process, due to a pressure enlargement of the canal for the optic nerve. Enlargement of the optic foramen may also be demonstrated, although it is, of course, necessary to take photographs with the head in a certain position to obtain an accurate record. The diagnosis of these cases should be relatively easy, although in the rare examples occurring in adults the diagnosis from suprasellar meningioma may be impossible. The following case history of a child still under observation illustrates some of the above points.

CASE III is one of glioma of the left optic nerve and chiasm. E.S., a female, aged eight years, when first seen at the Royal Alexandra Hospital for Children under the care of Dr. W. Vickers, gave a history of failing vision and a protrusion of the left eye for the past two or three years. Her mother stated that that eye had never seemed quite right, that she believed that the child had never seen properly with it, and that for four years before admission she developed a squint which was treated by glasses. There was also a history of irregular attacks of vomiting and of some pains in the head, both of which were relieved by liquorice powder given every week.

On examination in June, 1931, the left eyeball seemed a little larger, showed definite exophthalmos and swelling of the disk, and was apparently sightless. The child had a slight left-sided facial weakness and her mother stated that she was a little clumsy and awkward with her left hand and leg. There was no evidence of cutaneous neurofibromata. The diagnosis of simple orbital tumour was then made, and on June 16, 1931, the left eye was removed.

At the time of operation a tumour which was cystic in its anterior portion was cut across at the optic foramen, through which it apparently extended intracranially.

Recovery was uneventful, but since then the child gradually developed symptoms apparently due to an intracranial extension of the neoplasm. There were signs of involvement of the left motor pathway, with increased weakness and clumsiness of the left arm and leg, with a left extensor plantar reflex. In addition, the right eye showed early papillitis.

On examination the specimen shows involvement of the nerve head and of the optic nerve, with a peculiar tumour which was previously regarded as a neurofibromata, but which I believe to be one of the types of optic gliomata described by Verhoeff.

On re-examination on May 3, 1932, the child showed a definite temporal quadrantic defect in the visual field of the right eye, but little or no increase of swelling of the disk. In addition, there was a small neoplastic mass occupying the back of the left orbit, causing gradual dislocation of the artificial eye.

Radiographs showed definite enlargement of the right optic foramen and a pear-shaped protrusion of the sella forward under the anterior clinoid processes. (See Figure IV.)

Although this case shows an earlier involvement of the cerebral peduncles than one would expect, I believe the diagnosis of glioma of the optic nerve and chiasm with gradual extension of the neoplastic process into the interpeduncular space and consequent focal and general pressure symptoms is definitely established.

Suprasellar Meningioma.

For our present detailed and correlated knowledge of the pathology, symptomatology and effective operative treatment of suprasellar meningiomata we are almost entirely indebted to Holmes and Sargent[11] and to Cushing and his coworkers.[12] Like all meningiomata, which usually arise from arachnoid villi in the neighbourhood of venous sinuses, these arise from the tissue around the circular sinus in the region of the *tuberculum sellæ*, just over the chiasmatic sulcus. They become firmly attached to the dura, but unlike the meningiomata of the vault, have a lobulated mulberry-like surface, and do not give rise to a reactive hyperostosis. Also they are much less vascular and much slower in their growth than most meningiomata in other sites. As a rule the major portion of the tumour extends under the chiasm, which is elevated and compressed by it. Figure V, which is taken from Cushing, illustrates the mode of extension of these tumours.

Of course, as the growth becomes large, it tends to press upon the region of the infundibulum or the third ventricle and sometimes causes pressure atrophy of the clinoid processes. As a result various syndromes due to involvement of the hypothalamic nuclei, or compression hypopituitarism may develop as late manifestations.

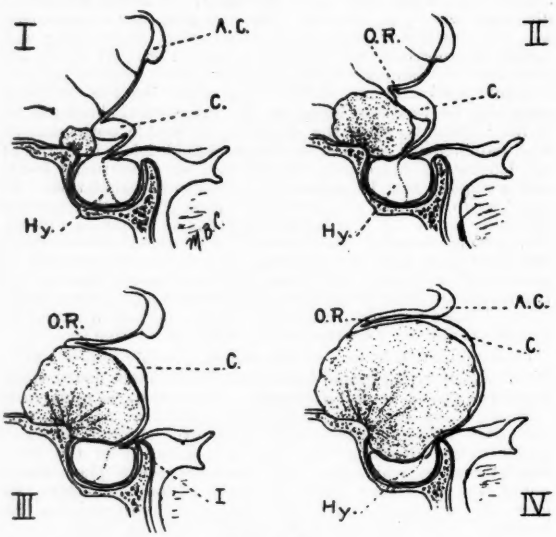

FIGURE V.

Showing mode of extension of a suprasellar meningioma. Series of drawings to illustrate on sagittal section in four stages the advancing deformation of chiasm and third ventricle: Hy. = hypophysis, C. = chiasm, O.R. = optic recess, A.C. = anterior commissure, I. = infundibulum. Number I is merely a presumptive initial stage. No. II is from an autopsy specimen and is possibly pre-symptomatic. Numbers III and IV represent the early and late stages producing the syndrome under discussion with tumours from about 10 to 18 grammes in weight. Anything larger than IV is likely to cause hydrocephalus. (After Cushing.)

Clinical Aspects.

Age.—Owing to their slow growth these tumours rarely manifest themselves before adult life, being, in fact, usually found in patients of middle age or over. In this respect they contrast markedly with the previously described suprasellar lesions.

Clinical Symptoms.—Typically pressure on the chiasm is from in front and below with corresponding involvement of the upper quadrant of the temporal fields, followed by slowly progressive optic atrophy. If the tumour happens to have its origin a little further forward, the pressure may come on the chiasm from above with resultant modification of the type of field defect. The usual finding, owing to the fact that one side is affected before the other, is optic atrophy in one eye with temporal hemianopia in its fellow. The sequence of development of the field defects may thus resemble very closely that found in the hypophyseal duct tumours. It is necessary to mention here the fact, emphasized so much by Cushing, that the pallor of the disks in these cases of gradually developing pressure may be so indefinite that its presence may be often a matter of opinion, even when the eye shows a very low visual acuity and grave defects in the fields of vision.

Papillœdema in these cases is very rare and can occur only in the very late stages, when owing to a rise in general intracranial pressure it is possible to have papillœdema superimposed on a primary optic

atrophy, provided always that the optic nerve sheaths are not completely blocked by the tumour.

General Pressure Phenomena.—General pressure phenomena are late manifestations, but in large growths they may, because of extension of the growth into the interpeduncular region, follow on more or less complete blindness. From the same cause a mild degree of hypopituitarism can occur; this, however, is never early and never a dominant feature of the symptomatology.

Radiological Appearances.—In the typical case the sella is normal in all respects, although some erosion of the clinoids and some slight pressure absorption of the sella may be noted in advanced cases. In a small proportion, approximately 10%, a calcareous nodule may be seen in the substance of the growth, just above the *tuberculum sellæ*.

The clinical picture of these tumours is therefore clear-cut, and as Cushing expresses it, it is the syndrome of primary optic atrophy and bitemporal field defects in a middle-aged person with a normal sella. Of all the lesions in this region this is the most amenable to surgical treatment and by using a special operative technique a permanent cure should be possible in the great majority of cases.

Tumours of the Anterior Part of the Third Ventricle.

Tumours of the anterior part of the third ventricle are of great interest, and a voluminous literature exists, for an exhaustive review of which the reader is referred to a paper by Fulton and Bailey.[13] They may originate in any portion of the ventricle. Histologically they are gliomata or ependymomata, and, owing to the fact that they may involve the hypothalamic nuclei of the infundibular area or give rise to internal hydrocephalus, pseudo-pituitary glandular syndromes are common.

Clinical Aspects.

Age.—From a study of the accurately recorded cases there seems to be no particular age incidence for these tumours.

Hypothalamic Symptoms.—There has been a good deal of confusion amongst clinicians concerning the explanation of many of the peculiar symptoms in lesions of the infundibular region. As emphasized by Walshe,[14] much of this "has arisen owing to the widespread omission of observers to draw any distinction between the normal function of a part and the symptoms resulting from lesions of that part—the well-known teaching of Hughlings Jackson on the dissolution of function being generally neglected". He criticizes particularly the looseness of expression which characterizes many commentaries which deal with the question of a hypothetical "sleep centre". However, our ideas with regard to the effect of lesions of this region are beginning to crystallize. It has been suggested by Cushing[15] that the posterior lobe of the pituitary and the hypothalamic nuclei constitute a neuro-hypophyseal mechanism and that various disorders of metabolism *et cetera* can result from lesions of either constituent. This seems to be undoubted, for many cases have been recorded in which obesity, *diabetes insipidus* and pathological drowsiness have occurred in which the pituitary was intact and apparently normal.

It is probable that further observations will show that some at least of the syndromes which have been regarded as due to hypopituitarism will be found in the future to be really hypothalamic in origin.

Fulton and Bailey in particular have emphasized the frequency of pathological somnolence associated with lesions of the infundibular area and quote many cases in which this symptom has occurred. This drowsiness is not to be confused with that due to internal hydrocephalus, so often seen in the late stage of practically any large intracranial tumour. These writers also maintain that destructive lesions of the *tuber cinereum* can give rise to *diabetes insipidus* with polydipsia, and quote the classical traumatic case of Maranon and Pintos[16] in support of this view.

In the same way, because dystrophy and adiposity are often a concomitant of the above symptoms, they believe, and this belief is borne out by some experimental evidence, that these symptoms are also due to lesions in the same site.

On the relationship between these syndromes and the function of the hypophysis opinion is still divided, the various viewpoints being put clearly by Cushing[17] in his recent work. There seems little doubt that we need no longer postulate a lesion of the posterior lobe of the pituitary as the cause of hypersomnia, adiposity or genital dystrophy, although such a lesion is probably a factor in the mechanism of *diabetes insipidus*.

If the infundibular syndrome appears early in the clinical chronology, it has a definitely localizing feature, a fact which up to the present has not been sufficiently realized.

In a later state of their history these tumours produce a great variety of focal and general effects. These include interference with the brain stem, so that pyramidal or cerebellar disease may be simulated, disturbances of heart regulation, incoordination *et cetera* of ocular movement, as well as mental and endocrinal disturbances. All these are very varied in their time of onset and no less than ten different syndromes have been postulated by various observers. Fulton and Bailey may be quoted as follows:

> There is no syndrome of the third ventricle, *per se*, but tumours immediately affecting this cavity may cause characteristic symptoms by pressure upon its walls and their contained nuclei and tracts. Among the well recognized syndromes due to lesions of these structures we may mention: (i) the infundibular syndrome (polyuria, adiposity); (ii) the syndrome of the central gray matter around the posterior end of the third ventricle and aqueduct of Sylvius (hypersomnia); (iii) the thalamic syndrome (central pain, painful hypesthesia); (iv) the extrapyramidal syndrome (bradykinesia, rigidity); (v) the decerebrate syndrome (hypertonicity, Magnus-de Kleijn reflexes); (vi) the syndrome of Parinaud (paralysis of conjugate vertical movements of the eyeballs); (vii) the syndrome of the body of Luys (hemichorea; *vide* Martin, 1927, Ewald, 1891); (viii) the hypopituitary syndrome (infantilism, hypotrichosis, lowered metabolism); (ix) the uncinate syndrome (olfactory and gustatory symptoms; *vide* Herzog, 1928).
>
> All of these syndromes are of as definite localizing value in cases of brain tumour as any other symptom when they occur precociously before the onset of pressure symptoms. The infundibular syndrome and the hypersomnia associated with lesions of the central gray matter are deserving of especial emphasis since, owing to imperfect understanding of their localizing value, they are seldom given the importance which they merit in localizing tumours of the brain.

The following case, although probably because of incomplete examination no chiasmal syndrome was observed, illustrates in some degree the endocrinal disturbance associated with these lesions. I am indebted to Dr. W. Vickers and Dr. F. Tidswell for permission to include it in this paper.

CASE IV.—J.N., male, aged four years, was first seen at the Royal Alexandra Hospital for Children in October, 1927, and was then regarded as suffering from disordered function of the pituitary gland. His mother stated then that his external genitals were abnormally large at birth, although he was an eight months baby. Pubic hair appeared at the age of three and a half years, and in the axilla at the age of four, while the hypertrophy of the external genitals rapidly increased. He was the second child of a family of three, the other children and the parents all being healthy. He had not walked till the age of two, and had always been mentally backward. At this time, in spite of the absence of any general disturbance of growth, of any abnormality of carbohydrate, water, or fat metabolism, of the absence of positive radiological findings, he was regarded as suffering from hyperpituitarism of a selective type. At the same time the possibility of the presence of both pineal and suprarenal neoplasms was discussed. At this time there was no evidence of increased intracranial tension.

In 1929, at the age of six, his genital hypertrophy had still further increased, while he had a well defined growth of hair on the upper lip. The lower limbs were of the adult pattern, being muscular and sinewy rather than soft. At about the same time his voice became deep toned, and he apparently became conscious of his abnormality, although he did not realize its significance. It is alleged that seminal emission occasionally occurred during the so-called fits that he suffered from. He tended to imitate men and to avoid children and females. From the age of four onward he was subject to peculiar fits, with marked emotional disturbance. He used to say that something went in his head and then he would grab a chair, the eyes both turned to the right, but at first he did not lose consciousness. These fits became worse—even to twitchings and rigidity of the body—and became more frequent as he grew older. At the age of six the "turns" became associated with outbursts of temper, the patient tending to kick, bite, scream and smash things, until he became unconscious. These "turns" would be up to half an hour in duration, but after they were over he became tractable and rapidly normal. He would fall asleep very readily after the attacks.

On examination at this stage there was no indubitable evidence of intracranial disease, and the neurological findings were normal. At no time was any papillœdema detected, the last examination of the fundus, however, being made at the age of six years; unfortunately no examination of the visual fields was ever carried out. The diagnosis at this time rested between pituitary or adrenal disease, but no certainty was reached.

At the age of eight he was again admitted to hospital with a story of having had six to twelve fits daily for a week and having been unconscious for two days. He died the day after admission.

Autopsy revealed a tumour of the floor of the third ventricle, projecting into the cavity of the ventricle and running forward to the infundibulum without causing any obstruction of the ventricular system. The *tuber cinereum* and the mammillary bodies were obliterated, while, although the optic chiasm was free, the optic tracts were lost in the posterior part of the growth. The tumour, which was histologically an astrocytoma, was perched on the basi-sphenoid, so that the right posterior clinoid process was flattened and deformed, but in spite of its position did not produce any hydrocephalus or any pressure on the bulbar region.

The pituitary gland was of normal size and appearance and careful microscopic examination revealed no structural abnormality. The same was true of the pineal body. As far as could be detected by macroscopic and microscopic examination, all the other endocrinal glands were normal.

It would seem that this interesting case adds yet another syndrome to those associated with tumours of this region. It is true that various observers have recorded cases in which emotional outbursts (the so-called "sham rage" syndromes) were a feature, but in none that I have been able to discover in the literature were they so marked as in this case. The remarkable hypertrophy of the external genitals with advanced development of the secondary sex characteristics are, I think unique, because, as a rule, lesions of the hypothalamic area of those which induce hypopituitarism lead to atrophic genital changes. It would seem, however, that for some reason or other the reverse effect may be produced by a tumour and this case illustrates the possibility of a neoplasm producing either destructive or irritative effects and so either increase or decrease of functional activity. It is also probable that this case would have shown defects of an homonymous nature in the visual fields, and from the situation of the optic tracts in the tumour it is conceivable that an accurate perimetric examination would have led to correct localization.

Meningiomata of the Olfactory Groove.

We are indebted to Kennedy[18] and Cushing[19] for most of our knowledge of the symptomatology of meningiomata of the olfactory groove. They arise from the arachnoid villi of the venous channels along the olfactory groove, gradually, however, coming to lie in the mid-line, and tending to extend back to reach the region of the sella. They grow very slowly, and owing to their position often reach a very large size before they produce definite symptoms. As they are benign tumours, they are, as Cushing has proved, amenable to surgical removal, with excellent chances of complete cure.

These tumours often involve one or both optic nerves or the chiasm, pressing down upon them from above, and may, either by direct pressure or by raising intracranial pressure, give rise to some interference with pituitary function. Although in this manner such a tumour may in some degree simulate pituitary disease, in reality the typical picture is unmistakable.

Clinical Aspects.

Following the general rule of the meningiomata, clinical symptoms are rarely produced by these slow-growing tumours before middle life.

Visual Defects.—The patient usually complains of failure of vision in one eye, which on examination shows primary optic atrophy. Not infrequently, owing to the frequent association with nasal symptoms, the condition is diagnosed as retrobulbar neuritis. The other eye shows varying field defects and gradual failure of vision often with papillœdema as the general intracranial pressure increases.

Anosmia.—Anosmia, either unilateral or bilateral, is present, although it is often missed by the patient and the clinician, being sometimes attributed to previous nasal operations. It is surprising how frequently these are performed in this type of case. There seems little doubt that unilateral anosmia, sometimes with subjective olfactory sensations, is in reality the first symptom followed by optic nerve involvement at a later stage.

General Effects.—General effects are due to pressure on the frontal lobe, so that these patients may show mental degeneration, becoming unreliable, euphoric,

or developing changes in disposition with loss of memory. Ultimately pressure on the infundibular region causes internal hydrocephalus, sometimes with symptoms of mild hypopituitarism.

Radiographical Appearances. — Radiological examination may reveal erosion of the sphenoidal ridge, with a medial patch of calcification above the region of the *crista galli*, and not infrequently in the advanced cases, some pressure enlargement of the sella.

These tumours give a clear-cut syndrome, providing that an accurate chronological record is obtainable. The sequence of events should be: Unilateral anosmia, often unobserved; homolateral failure of vision due to optic atrophy; complete anosmia, often noted; contralateral hemianopia and papillœdema; mental changes from compression of the frontal lobe, in a middle-aged person, with, as a rule, a normal sella.

Rarer Lesions.

In addition to the suprasellar lesions described above, there are several rare types of tumour which can occur in this region. They are chordomata, teratomata, cholesteatomata, simple cysts of the remnants of the original cavity of Rathke's pouch, and tumours of the chorioid plexus. These must be regarded as pathological curiosities, and are clinically indistinguishable from the other suprasellar tumours.

Two other conditions deserve special mention—aneurysm of the arteries of the circle of Willis and chronic arachnoiditis of the basal cistern. These two conditions are now well known as rare sources of error in diagnosis, and have been described by Symonds,[20] Harris,[21] Craig and Lillie,[22] Cushing,[23] Horrax,[24] and others.

Distant Lesions.

It has long been recognized that a high intracranial tension which may be induced by any tumour, but particularly by those of the posterior fossa, may lead to symptoms of hypopituitarism, and give rise to alterations in the contour of the *sella turcica*. It has been suggested by Cushing and Goetsch[25] that cerebro-spinal fluid stasis produces in effect an experimental obstruction of the infundibular stalk, so that the secretion of the posterior pituitary lobe may not be absorbed. As a result most of the symptoms shown are those of hypopituitarism, taking the form of adiposity (it is well known how well nourished most cerebral tumour patients remain even in the late stages) amenorrhœa, anaphrodisia, lowered metabolism, and interference with carbohydrate metabolism. Some of these patients may apparently show a mixed glandular syndrome or even hyperpituitarism.

It is common to find the sella somewhat enlarged from pressure atrophy, with some flattening of the clinoid processes, while the skull almost invariably shows some degree of convolutional atrophy of the inner table.

The tumours which most commonly give these simulations of pituitary disease are cerebellar astrocytomata of comparatively long standing. Grant[26] and Bailey[27] have recently emphasized the confusion that can exist in the diagnosis between cerebellar tumours and tumours of the pituitary region. Ophthalmological examination is of the greatest value, as these cerebellar lessions produce, as a rule, bilateral papillœdema without hemianopia, although there may be some constriction of the visual fields—a very different picture to that found in pituitary or suprasellar lesions, and one which, even in the absence of any localizing signs, should lead to a careful review of the case.

Conclusions.

A study of cases such as the foregoing with a review of the literature, emphasizes the great importance of an accurate ophthalmological study of each case. In all of them a careful examination of the eye grounds, and particularly of the fields of vision with special attention to early temporal defects, as determined by examination with small disks, is essential for success. Success or failure in the diagnosis of all lesions that involve the optic chiasm, depends entirely on perimetry, just as post-operative examination of the disks determines the degree of success or failure of surgical intervention. In the same way it is the ophthalmologist who must determine in a great proportion of cases whether or not an intracranial operation should be attempted or whether some other procedure should be adopted. Until this is generally recognized by physicians, surgeons and ophthalmologists, and full cooperation is obtained, full success in the diagnosis and treatment of these lesions cannot be achieved.

In a similar manner, although to a lesser extent, the neurosurgeon is dependent on skilled radiographical interpretation. This necessitates increasing experience with the normal variations, an appreciation of some of the difficulties of the problem before us, and active cooperation towards a common end.

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Reports of Cases.

UTERINE GAS GANGRENE: THREE CASES OF RECOVERY.[1]

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THE condition of infection with *Bacillus welchii* of the retained products in cases of incomplete abortion is not common, and in many of the recorded cases fatal results have ensued. The patients here reported all recovered, albeit after a stormy passage, and were quite well when last hard from, eighteen months after their illnesses.

We may lay down an axiom that the presence of *Bacillus welchii* infection in the uterine contents allows the presumption, beyond peradventure, of mechanical interference in some form with the normal conception. This fact was established in two of the cases and was practically established in the third case, in spite of strenuous denials.

One of the patients was extraordinary in that frank septicæmia was present on admission to the hospital, *Bacillus welchii* being found on blood culture. And this serious condition was attributed to a splinter which had been run into her thumb and successfully removed ten days before. A gynæcological opinion was not sought till she had been in hospital forty-eight hours, and then only for "menorrhagia". The prevarications of this patient were extremely facile.

The clinical aspect of these patients is distressing, compared with that of ordinary patients suffering from incomplete abortion. There is extreme pallor and marked restlessness is present, simulating at times the general appearance associated with ruptured ectopic gestations. Pain is not an outstanding feature. Probably the most notable point on clinical examination is the extreme fall in systolic blood pressure. This sign, in conjunction with the physical signs of incomplete miscarriage, made the diagnosis of probable *Bacillus welchii* infection possible in the second and third cases and expedited the commencement of serological methods of treatment.

Gas gangrene antiserum was administered to all these patients in what appeared to be adequate doses. In the first two cases there was certainly no dramatic recovery following the administrtion of the serum. The patients improved slowly and steadily, but one naturally wonders if this happy result might not have been attained without serum therapy.

Our third patient received serum in similar doses, but in addition was subjected to intrauterine instillation of glycerine thrice daily—a procedure which involves very little movement, even in a patient who is very ill. The result was certainly brilliant compared with that of our first two cases; toxæmia ceased at once, pyrexia rapidly subsided, and the patient was discharged ten days after admission to hospital, apparently quite well.

[1] Read at a meeting of the Section of Obstetrics and Gynæcology of the Victorian Branch of the British Medical Association, September, 1932.

Blood transfusion is usually resorted to in view of the extreme anæmia in these patients. Once the infection is overcome, however, blood regeneration is extremely rapid and complete.

The operation of emptying the uterus in these cases is usually complicated by really severe hæmorrhage; in fact, a good deal of the retraction of uterine muscle fibres seems to be in abeyance. The usual cause of continuance of bleeding in curettage for miscarriage is a fragment of placental tissue overlooked by the operator and retained *in utero*. With these patients, in spite of all care to see that the uterine cavity is indeed empty (and this is proven by no further fragments being extruded at a later date), hæmorrhage continues and is best controlled by a plug of iodine gauze removed in twelve hours.

The histories of three cases of gas gangrene infection of retained secundines are attached.

Case I.

Mrs. O.M., aged thirty-two years, was seen on November 22, 1930, by her physician, who was called in on account of a fainting attack. She had been "off colour" for three to four days, and was about four months pregnant. Any attempt at abortion was strongly denied. The spleen was found to be greatly enlarged and the uterine enlargement corresponded to the date of amenorrhœa. Nevertheless her physician considered that some gynæcological interference had occurred. She was admitted to the medical wards of the Alfred Hospital on November 23, 1930, with a provisional diagnosis of splenic anæmia.

On admission she complained of debility with loss of weight for three weeks. The past history was clear; the spleen had been known to be enlarged for fourteen years. She had had two children and no miscarriages, the youngest child being four years old.

On examination the patient was very pale and restless. The temperature was 37·5° C. (99·6° F.), the pulse rate was 132, and respirations numbered 28 in the minute. The systolic blood pressure was 110 and the diastolic pressure 70 millimetres of mercury; the pulse was thready. Examination of the heart and lungs revealed no abnormality. Abdominal examination showed no undue tenderness; the uterus was enlarged almost to the umbilicus and the spleen was much enlarged, the edge being easily palpable midway between the umbilicus and the costal margin. The fœtal heart could not be heard. *Per vaginam* the cervix was found to be lacerated, but closed, and there was no loss of blood. Urinary examination revealed no albumin or sugar, but a trace of acetone. Blood examination revealed 1,660,000 erythrocytes per cubic millimetre, 10,000 leucocytes per cubic millimetre, and a hæmoglobin value of 25%.

On November 24 she received a transfusion of 600 cubic centimetres of citrated blood. At midnight abortion of a four and a half months fœtus occurred, the membranes and placenta being retained *in utero*. On November 25 the secundines were still retained and a gynæcological opinion was sought.

The condition at this time was poor—the uterus was still much enlarged, the external os was patulous and choked with *débris*, and the patient was losing a small quantity of blood. After a further transfusion of 300 cubic centimetres of citrated blood the retained products were removed with ovum forceps and the blunt curette under general anæsthesia induced by ethylene. The hæmorrhage was excessive and was controlled with a plug of iodine gauze, which was removed in twelve hours.

The blood taken at this time yielded *Bacillus welchii* on incubation, and there were innumerable *Bacilli welchii* in the decidual fragments.

After desensitizing the patient treatment with gas gangrene antiserum was energetically adopted; 20,000 units of serum were given twice a day, intravenously with saline solution for the first two doses, and then intramuscularly. The reaction was not entirely satisfactory and the patient, on the eighth day after operation, still had a swinging temperature up to 38·3° C. (101° F.). Attempted blood culture at this stage yielded no growth. Œdema of the extremities now developed and the serum dosage was lessened to 10,000 units a day, given intramuscularly. Two days later frank jaundice appeared and

lasted for nine days. The serum dosage was entirely remitted during this time.

The general condition improved as the jaundice faded, and the patient left hospital, well, four weeks after her curettage.

The total dosage of serum in this case was 280,000 units.

Case II.

Mrs. M., aged forty-three years, was admitted to the medical wards of the Alfred Hospital on January 9, 1931, complaining of progressive malaise following a splinter in the thumb fourteen days before. Her previous history was clear. She had had two children, the youngest being ten years old and she denied any miscarriages. She stated that she ran a splinter into her right thumb fourteen days before admission to hospital. Four days later there had been progressive onset of vomiting, shivering, sweating, thirst, anorexia, and generalized abdominal pain, but no diarrhœa. The next day her physician had removed the splinter from her thumb and had noted a tender gland in the axilla.

Her condition had become progressively worse, weakness being a salient feature. Vomiting increased, and she was exhausted on her admission to the hospital. The bowels were costive, the micturition normal, and she claimed to be menstruating at the time of admission.

On examination the temperature was 39·2° C. (102·6° F.), the pulse rate was 132 and respirations numbered 28 per minute. She was very pale and distressed and extremely restless. Examination of the heart and lungs revealed no abnormality. The systolic blood pressure was 90 and the diastolic pressure 50 millimetres of mercury; the pulse was very soft in volume. Abdominal examination revealed no undue tenderness or rigidity; the liver and spleen were not palpable and there were no petechiæ.

No abnormality was discovered in the central nervous system.

Treatment was instituted, consisting of free administration of fluids and stimulants, blood was taken for culture and preparation was made for blood transfusion.

Four hours after the blood was taken for culture, a report was received from the laboratory that gas bubbles formed in the inoculum. After desensitization, treatment was commenced by giving 20,000 units of gas gangrene antiserum with 200 cubic centimetres of saline solution intravenously.

Serum therapy was continued during the next two days, during which 80,000 units were administered. The patient's condition was certainly no better; nausea and vomiting were still marked features. The erythrocyte count was 3,740,000 per cubic millimetre. Menorrhagia was pronounced and the patient was very pale and restless.

Gynæcological opinion was sought and the uterus was found to be enlarged, the os patulous and choked with clot. The condition was diagnosed as *Bacillus welchii* infection of incomplete abortion. A blood transfusion of 600 cubic centimetres of citrated blood was given and the uterus was evacuated with ovum forceps and the blunt curette. The hæmorrhage was very troublesome and had to be controlled by an iodine gauze plug. The smears made from the decomposing placental fragments showed innumerable *Bacilli welchii*.

On the removal of the uterine plugging next day the general condition markedly improved. Gas gangrene antiserum was continued in doses of 10,000 units every four hours for the next two days. On the fourth day after operation the attempts at blood culture yielded no growth.

Satisfactory progress now ensued, except for a fairly severe serum rash, which responded to treatment. The patient went home, well, three weeks after the curettage. The total dosage of serum in this case was 220,000 units.

Case III.

Mrs. A.V., aged thirty-one years, was admitted to the Alfred Hospital on March 15, 1931, complaining of intense lower abdominal pain for twelve hours, having noticed hæmorrhage *per vaginam* for the previous three days.

She had had two children, the youngest being ten years old, and had had a pelvic abscess following appendicectomy twelve months before.

The patient stated that three months before admission to hospital she had had a normal period. The next two periods were very scanty, the last period being due a week before admission. For three weeks she had suffered from nausea, but no vomiting. Three days before admission there had been a severe hæmorrhage, with many clots; in the last twelve hours agonizing pain in the lower part of the abdomen had commenced and persisted in spite of administration of morphine.

The bowels had been very costive and there had been no abnormality in micturition.

On examination the patient was very pale and restless, with cold, clammy skin. The temperature was 39·7° C. (103·5° F.), the pulse rate 108 and respiratory rate 36. The systolic blood pressure was 72 and diastolic pressure 45 millimetres of mercury. Examination of the heart and lungs revealed no abnormality.

The abdomen was lax, with some tenderness in the suprapubic area. Vaginal examination showed the uterus to be about three times the normal size, the os patulous and choked with clot, while both fornices were exquisitely tender.

Diagnosis of infection with *Bacillus welchii* was made.

Under general anæsthesia induced by ethylene, immediate evacuation of the uterus by means of ovum forceps and the blunt curette was undertaken. There was profuse hæmorrhage during the operation, which had to be controlled by packing with iodine gauze. Blood culture taken at this time was positive for *Bacillus welchii*, and smears from the tissue removed contained many of the organisms also. Transfusion of 600 cubic centimetres of citrated blood from the husband was undertaken as soon as the operation was completed, and 20,000 units of gas gangrene antiserum were administered intramuscularly after preliminary desensitization.

The gauze plugging was removed in twelve hours and intrauterine instillation of 50 cubic centimetres of warm glycerine was instituted thrice daily; 20,000 units of gas gangrene antiserum were also administered daily. After four days the general condition was so much improved that both glycerine and serum therapy were discontinued.

Douching with permanganate of potash, one in eight thousand, was continued for another three days, the patient's condition being eminently satisfactory. The systolic blood pressure was 110 and the diastolic pressure 80 millimetres of mercury. She left the hospital, well, ten days after operation. The total serum dosage in this case was 100,000 units.

CEREBELLAR HÆMORRHAGE.

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A FEW weeks ago I watched a man competing vigorously in a wood-chopping contest and was most surprised when I was called by the coroner a few days later to investigate the cause of his sudden death.

The history obtained from relatives was that he was forty-three years of age. He was a sleeper-cutter. He had been at work each day previously and had his normal breakfast at 7.30 a.m., commencing work at 8.30 a.m. He had rolled a billet of wood into position and was commencing to square it when he had a violet fit of coughing, which lasted four to five minutes. His mates went to his assistance and found him on his elbows and knees with his head in his hands. He had the premonition of death and was quite helpless. He complained of intense pain in the back of his neck and occipital region and vomited frequently. He was taken to his home by motor lorry and died at 3.30 p.m. The vomiting, pain in the back of his neck and head, weakness, and the premonition of death were his only symptoms so far as I could learn. He was quite conscious to within a few minutes of his death and could move his arms and legs.

Post mortem examination revealed a tall, thin man with no evidence of any external injury. The alimentary tract was clear. The stomach walls were healthy and the mucosa showed no signs of irritation. In the kidneys there

was no evidence of nephritis or of arteriosclerosis. The lungs were mottled and spongy; there was no fibrosis; there were no adhesions in the pleura. The heart appeared normal in size; the valves and muscle were healthy; the coronary arteries were clear and open; the aorta was clear; there was no blood clot in the heart. On examination of the brain the hemispheres and medulla appeared normal. Blood-stained fluid was found in the ventricles. In the cerebellum there was a large hæmorrhage, mainly in the left half and partly in the right, opening into the fourth ventricle. No ruptured vessel was found, and the circle of Willis was healthy.

I gave the cause of death as cerebellar hæmorrhage into the fourth ventricle, though I could see no cause for the hæmorrhage.

Reviews.

MODERN GENERAL ANÆSTHESIA.

"MODERN GENERAL ANÆSTHESIA", by Dr. James G. Poe, is an elementary book intended apparently for those who are slow in the uptake.[1] To secure emphasis, often of unimportant points, heavy type is scattered about and every now and again summaries are inserted. The author's style is prolix and didactic and single sentence paragraphs are numerous. All the general anæsthetics in use at the present day are more or less tediously discussed. The author's condemnation of chloroform is unrestrained, but he is tempted beyond the limits of proved fact when, for instance, he alleges that its administration to ease the pangs of labour may years afterwards adversely turn the scale should the victim contract pneumonia. He attempts to describe the administration of chloroform though it is apparent that he knows very little about the subject. In the chapter on "Carbon Dioxide and Inhalation Anesthesia" we are informed that "The principal results of acapnia are depression (slow and shallow) or suspended respiration, apnea, diminished peristalsis, and reduction of blood-pressure through vasomotor influences (circulatory shock)". And a little further on: "If the acapnia should be in the extreme and respiration is prolonged from some cause beyond safe limits, as exhibited by slight cyanosis, artificial respiration should be resorted to." We find ourselves unable to translate these remarks into English. Under "Care of Patients" we are warned that "The Fowler position is to be avoided, unless it is necessary for drainage, as this position increases the danger of the development of post-operative thrombosis and embolism". No proof is offered in support of this contention. As, however, we do not see the Fowler position adopted "unless it is necessary for drainage", we need not argue the point. Yet this book has reached a second edition.

CLINICAL PÆDIATRICS.

THOSE to whom the diseases of children are of more than ordinary interest will find much to enjoy and much to ponder over in "Clinical Notes on Disorders of Childhood", by D. W. Winnicott.[2] In his preface the author states that the book proceeds from the heart of a clinician rather than the brain of a literary student. Though we cannot recommend the book to the student, it must be of interest to the pædiatrist with sufficient clinical experience to grasp without misconception the portent of the author's statements.

> The apex beat in a child is commonly felt half an inch outside the nipple line in the fourth or fifth space, and this finding alone, even with the child lying in bed, cannot be used as an argument in favour of carditis or hypertrophy.
>
> Even if a child, during examination, is found to have the apex beat more than half an inch outside the nipple line it is by no means certain that the heart is diseased. For the common cause is anxiety—and it is very common for a child to be apprehensive while being examined.
>
> If anxious children whose hearts are thus dilated when they are being examined are allowed to lead active lives they do not develop heart disease. In fact, dilatation due to active carditis is much less frequent than that which is simply part of the vaso-motor changes resulting from anxiety.

Statements such as these, strictly accurate though they be, cannot be put into practice dissociated from a thorough understanding of the earliest signs and symptoms of rheumatic carditis. This is dealt with fully and clearly. However, apart from the rheumatic diathesis and its associated carditis, the remainder of the book is devoted to consideration of various aspects of the nervous system of the child.

It would seem that the author's worry is that a prolonged period of rest in bed, ordered to a child suffering from anxiety and not rheumatic carditis, will cause irreparable harm, but that an early heart lesion must not be overlooked because of this. The majority of us are apt to overlook the harm to the nervous system of the child in our anxiety to protect the heart. It is well that we should read such a book. Some will not be prepared to accept all the vagaries attributed to the nervous system, though most will derive interest from the views expressed.

The chapters on anxiety, micturition disturbances, masturbation and stuttering, subjects usually avoided in text books, are of value. The author's views on these subjects are influenced by his association with Professor Freud and give evidence of a leaning towards the psycho-analytical methods of treatment.

Notes on Books, Current Journals and New Appliances.

A TALE OF THE SOUTH SEAS.

"WHITE MAN, BROWN WOMAN" is the life story of a trader in the southern seas.[1] The author, J. L. Richards, has lived a remarkable life; Stuart Gurr is associated with him as author of the book. We presume that Mr. Gurr has done the writing; the book is well written and holds the attention. Native life and customs are vividly portrayed and the islands and their beauty are well described. It would be easy to find fault with the author's knowledge of matters nautical. For example, a main sheet cannot be torn to ribbons, for a sheet is a rope, not a sail. These technical errors, however, do not detract from the tale as a tale.

The author married a native who bore him several children; he tells of his experiences frankly and shows once again that union of white men with native women leads to disaster. The title of the book and the printing on the paper wrapper of some words from the preface, "I have lived a life beyond the pale", are presumably chosen to attract a certain class of reader. This is a pity. The book deserves a better fate.

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[1] "Modern General Anæsthesia: A Practical Handbook," by J. G. Poe, M.D.; Second Edition; 1932. Philadelphia: F. A. Davis Company. Demy 8vo., pp. 231, with illustrations. Price: $2.50 net.

[2] "Clinical Notes on Disorders of Childhood" (Practitioner's Aid Series), by D. W. Winnicott, M.A., M.R.C.P.; 1931. London: William Heinemann (Medical Books) Limited. Foolscap 4to., pp. 224. Price 10s. 6d. net.

[1] "White Man, Brown Woman: The Life Story of a Trader in the South Seas", by T. L. Richards and S. Gurr; 1932. Crown 8vo., pp. 281. Price: 6s. 6d. net.

The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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PHYSICAL FITNESS IN SCHOOL PUPILS.

AMONGST the most important of the many activities of the practitioners of preventive medicine should be an adequate survey of the physical state of boys and girls at school. Less dramatic and obvious than the wholesale inoculation of communities against disease or the defensive fight against invading pestilence, but vital to the health of a nation is the maintaining and fostering of a high standard of physical well-being in the young. The strain of school years is well known. The earlier years are usually those of common epidemic disease, which up to the present time we seem forced to accept, reluctantly, as well-nigh inevitable. But the later school years, including those at secondary schools and universities, impose fresh strains. W. R. P. Emerson, in an article on physical unfitness in school pupils, puts the matter well when he remarks that the school standard *plus* the extra-curricular activities of the student mean a hectic life for pupil and teacher alike.[1]

[1] *The American Journal of Diseases of Children*, September, 1932.

In this country, as in others, the importance of keeping a check upon the nutrition standards of school pupils is well recognized, and some effort is being made to correct divergences therefrom. More belatedly attention has been drawn to the physical state of university students. In both fields there is wide scope for useful work, but certain difficulties lie in the way. Emerson points out that disease is often recognized, but not its predisposing factors, and among the latter he places faulty habits of work and play, overactivity during the period of rapid growth, and hurried and improper eating. It may be remarked also that it is one thing to bring a child's remediable defects to the notice of the parents, but it is quite a different matter to secure for the child the medical attention he needs. In other words, the connecting link between the discovery of physical anomalies and their removal is difficult to forge. It may be claimed that a lowered physical standard in the child is related in part at least to the social status and opportunities of his family. That this is not necessarily true is shown by Emerson, who found that a greater degree of unfitness and a higher percentage of faulty health habits were actually present in those private schools equipped with gymnasia, adequate playgrounds, medical staffs, and special instructors governing the physical training and the dietary of the students. Perhaps some selective factor operates here, for possibly in some cases the parents of physically backward children make an effort to send them to a school with (apparently, at least) special facilities for physical training. But even if this be the case, it is really no excuse in many instances, for it brings us back to the statement made by this author, that defects are recognized but their predisposing causes are not.

Emerson presents in this study numbers of tabulated statements in which a statistical basis is given for his conclusions. Several interesting points emerge. That marked aberrations from the norm of weight for a given age are important will be readily admitted. He finds that students over the weight standard most commonly present digestive disturbances, and that the cause for departure from

health is most frequently found in the alimentary system. Students who are under-nourished most often fall victims to respiratory disease. The great importance of the maintenance of a standard weight for age ratio in children is clearly seen in this inquiry. That there will be variations from the average is obvious, even amongst healthy children, and individual allowances must be made. But how many parents, how many educational authorities, how many medical practitioners really heed this significant yet simple index of health? In fact, one is tempted to go further and to remark that since the importance of the weight for age ratio is so obvious there are in this matter, as in others, perhaps two classes of people who can overlook the obvious—the unintelligent who pass it by, and those so intelligent that they are irritated by it and thus fail to heed its warning. Emerson rightly states that it is an "erroneous idea that if a person is not ill he is well". Many school teachers are imbued with this error, and many more parents.

Those interested in medical work in schools will probably find in this article only a confirmation of their own views, reduced to figures. But an appeal should be made to a wider circle, and all practitioners of medicine could with advantage take to heart another of the author's aphorisms: "Medical training should be as efficient in the diagnosis of health as in that of disease."

THE NEW SOUTH WALES MEDICAL BOARD.

In another place in this issue we publish a letter from Dr. G. H. Abbott, to which the attention of our readers is directed. The public of New South Wales was informed recently in the daily Press that a new Medical Board had been appointed. Some newspapers displayed enormous headlines to this effect. The casual reader gathered that the Minister, Mr. R. W. D. Weaver, had adopted a policy for the retirement of members of the Board when they reached the age of sixty-five years. Dr. Abbott's letter will show how the Minister chose to deal with men who have given many years of honourable and gratuitous service to the State. No one will gainsay the Minister's right to exercise his discretion in regard to the Medical Board, but many people will resent his remarkable lack of courtesy. We take this opportunity of expressing to Dr. Edward J. Jenkins, Dr. W. H. Crago, Dr. G. H. Abbott, Dr. J. Adam Dick, Dr. W. G. Armstrong, and Dr. A. Murray Will the appreciation of the medical profession throughout New South Wales and the Commonwealth for their unselfish devotion to duties which must at times have been onerous and exacting.

Current Comment.

BISMUTH AND SALYRGAN AS DIURETICS.

DIURETICS are notoriously uncertain in action, and in conditions of anasarca and ascites, when it is necessary to remove quantities of fluid from the body, they often fail disconcertingly. Further, many diuretics are irritating to the kidneys, so that when they are most required, it is unwise to use them. There is a constant search for some new or different diuretic which will be more certain in action and which will not be attended with dangerous effects. A xanthine compound, "Euphyllin", has been lauded. "Novasurol" ("Merbaphen") has been in use for some time, and some years ago was studied by C. G. Lambie. Its claims for inclusion in the new pharmacopœia were considered, but finally it was rejected. Theophylline ("Theocine") sodium acetate has been included in the pharmacopœia, and juniper and scoparium have been omitted.

A. B. Stockton extols the efficacy of bismuth sodium tartrate as a diuretic and anti-œdemic.[1] His results were obtained from observations on fifteen patients. Two of these patients had normal cardio-vascular systems; three had portal cirrhosis and ascites; nine had cardiac decompensation and œdema, and one had degenerative Bright's disease with anasarca. The patients were kept in bed for a control period without medication. Bismuth was injected when urinary output remained constant. A constant intake of fluid and sodium chloride was maintained. Most of the patients had received medication with other diuretics without benefit. Only four had bismuth as the initial diuretic. In ten of the patients there was marked diuresis. In fourteen out of eighteen trials definite diuresis resulted. In the others diuresis was absent or slight. As occurs with other diuretics, some patients manifested diuresis after one injection, but not after other injections given before or subsequently. Most patients responded to the first diuretic agent with

[1] *Archives of Internal Medicine*, July, 1932.

increase of urine, but not after the next, unless an interval of two to seven days had elapsed. The maximum percentage diuresis was obtained by comparing the mean daily output of urine with the peak of daily output after bismuth injection. The percentage change in the total diuresis was indicated by the difference between the volume of urine excreted during three days before and three days after injection of bismuth. A definite and considerable increase in the output of urine was noted and the diuretic action was well sustained. There was an absence of secondary oliguria. In most of the patients there was increased excretion of chlorides in the urine. The changes in the chlorides were in the same general direction as those in the diuresis, but the magnitude of the increase was less. The increase in urinary output would account for increased excretion of chlorides. Active diuresis following administration of bismuth was always accompanied by increase in blood chlorides. The changes in the blood chlorides closely paralleled changes in diuresis and in urinary chlorides, indicating the tissues as the source of the extra chlorides. There was definite increase in the urinary urea in all the patients, and it would seem that the increase in the urea was due to the increased diuresis. In six instances there was no change in blood urea following bismuth administration, and in one patient there was a fall of blood urea with clinical improvement. Loss in body weight was marked in all œdematous patients who showed active diuresis. Bismuth sodium tartrate caused diuretic and anti-œdemic actions in 73% of the œdematous patients observed. The œdema fluid was removed at a slower rate than with other diuretics, but as the diuresis continued for several days, more fluid was removed than with other diuretics. The most significant changes were the simultaneous increase in the chlorides of the blood and urine during the increased diuresis, indicating a general tissue action of bismuth. The changes in the chlorides of the blood and urine agree closely with those observed in rabbits treated with bismuth. The chloride in the blood was lower in œdematous patients than in normal subjects. This agrees with the view that salt is retained in the tissues during œdema and accounts for the low concentration of salt in the blood. The practical advantages claimed for bismuth are that the diuresis is well sustained and that much fluid is removed. No nausea and vomiting are caused, and danger of metallic poisoning (stomatitis and renal injury) is much less than with "Merbaphen" or "Salyrgan". Its slow onset of action is a disadvantage. It is not so spectacular as "Novasurol" or "Salyrgan". Like other diuretics, it has a variable action.

L. Tarr and S. Jacobson contribute a eulogy of "Salyrgan" ("Mersalyl").[1] They assert that it is devoid of the harmful potentialities of "Novasurol". In more than three thousand injections of "Salyrgan" there were only isolated instances of harmful reactions. Thirty patients were particularly studied, most of whom had signs of severe congestive heart failure. "Salyrgan" is a complex organic mercurial compound. The toxic action of mercury is both immediate and remote. Only a negligible number of reactions could be attributed to the mercury in "Salyrgan", which was withheld only in the treatment of those showing severe renal change. Albuminuria and casts were not considered contra-indications. In two patients with impaired renal function a mild stomatitis followed a single injection. Only one patient, aged five, showed a renal lesion suggestive of mercury poisoning. Difficulties arise when seeking the finer anatomical criteria of hydrargyrism in patients who have long suffered from chronic passive congestion of the viscera. In older patients arteriosclerosis or arteriolosclerosis with destruction of renal parenchyma makes the presence of changes due to mild hydrargyrism difficult to determine. The authors considered that only severe necrosis of the tubular epithelium or calcareous deposits justified a diagnosis of mercurial poisoning. This attitude is convenient and helps their special pleading, but is scarcely logical. In a few instances the colonic mucosa was congested and œdematous, but no severe lesions were observed and ulceration did not occur. Conclusions as to the toxicity of mercurial diuretics based on animal experiments can be applied to man only with caution. "Salyrgan" was found by K. O. Möller to be as toxic to rabbits as was "Novasurol", causing albuminuria, hæmaturia and renal casts. Repeated injection caused chronic nephritis. In dogs he found no evidence of renal irritation and believed that man resembled the dog in his behaviour to the drug. It would be interesting to know why this conclusion was reached. E. Fourneau and K. I. Melville found that the intake of fluid was an important factor in determining the toxic dose of mercurial diuretics in rabbits and probably in other animals. Doses were no longer fatal, if attended by the administration of much water. These observers found the "Novasurol" group of mercurial diuretics to be less toxic to rabbits than the "Salyrgan" group.

In spite of the eulogy of Tarr and Jacobson concerning "Salyrgan", their conclusions are not convincing. Both "Salyrgan" and "Novasurol" should be used only in persistent cases of œdema, and then only when there is no, or but little, nitrogenus retention. If there be actual destruction of secretory tissue, these drugs may be extremely harmful and may rapidly set up mercury poisoning, with stomatitis, diarrhœa and aggravated renal disorder. Bismuth as a diuretic is not promising. Its use in syphilis has not been free from toxic sequelæ in the organs of its excretion. Further, the use of diuretics which require intramuscular or intravenous administration is not always practicable. If mercury be indicated, Guy's diuretic pill, of hallowed memory, is still efficacious if appropriately used. Even with "Salyrgan" and "Novasurol" the adjuvant action of ammonium chloride or ammonium nitrate is often necessary.

[1] *Archives of Internal Medicine*, July, 1932.

Abstracts from Current Medical Literature.

THERAPEUTICS.

The Human Heart in Anæsthesia.

I. G. W. HILL (*Edinburgh Medical Journal*, September, 1932) discusses electrocardiograms taken from forty-one patients while under anæsthesia. In most cases the records were made during induction and throughout operation. Connexions to the string galvanometer, about 150 yards from the operating theatre, were made by overhead wires. Usually all three leads were available. Special points in the operative procedure were notified by signal. The anæsthetics used were chloroform, ether (but only after induction by some other anæsthetic), gas and oxygen, ethyl chloride and "Avertin". In eight of sixteen cases in which chloroform was used for induction gross disturbances of cardiac action were produced. The commonest abnormality (seven cases) was the production of multiple ventricular extrasystoles. In one case paroxysms of supraventricular tachycardia alternated with the extrasystolic disturbance. In one case an exaggerated "vagal" arrhythmia appeared, with phasic variation in rate and in the *P* waves. In general the disturbances were a feature of the period of induction and passed off with deepening anæsthesia. They were not related to operative procedures other than direct stimulation of the vagus. They were increased by struggling on the part of the patient. They usually occurred in healthy hearts, were not accompanied by any marked clinical symptoms and there was no interference with the course of the anæsthesia. In the case of other anæsthetics only minor abnormalities appeared, such as fluctuations in rate, abolition or inversion of the *P* waves, nodal rhythm with negative *P-R* intervals, or aberration of ventricular complexes. A conspicuous feature of gas and oxygen anæsthesia was the occurrence of marked but transient slowing of the heart early in induction; this was not due to auriculo-ventricular heart block. In some cases slowing of the heart followed traction on the carotid vessels or interference with the recurrent laryngeal nerves. But in general, operative manipulations (opening of the peritoneum, ligature of cystic vessels and duct, dilatation of the anal sphincter, thyreoidectomy) were without influence on the heart. The disturbances noted in the case of chloroform are compared with the pre-fibrillation phase observed in the cat under light chloroform anæsthesia. In two cases direct stimulation of the exposed vagus nerve in patients fully anæsthetized by chloroform was followed by the appearance of extrasystoles without effect on the heart rate. In those cases in which "Avertin" had been given half an hour before gas and oxygen, the records tended to show more abnormality than under gas and oxygen alone. For example, one case showed wide fluctuations in rate (150, 60, 125, 110, 160) with variations in the amplitude of the *P* wave, frequent ventricular extrasystoles and variation in the *Q R S* complexes. Ethyl chloride was used in four cases for induction, and in two of these records were taken during that period. In neither was there any electrocardiographic change beyond one of rate. The literature is fully discussed and it is pointed out that graphic records have been published in comparatively few instances.

Makaradhwaja.

R. N. CHOPRA AND B. MUKHERJEE (*The Indian Medical Gazette*, August, 1932) have investigated the properties of *makaradhwaja*, a preparation of sulphide of mercury employed by ayurvedic practitioners in India. In the preparation of *makaradhwaja* eight parts of pure mercury and one part of gold leaf are mixed to form an amalgam; sixteen parts of sublimed sulphur are added. The mixture is rubbed to a fine powder with a stone pestle in a stone mortar. The powder, known as *kajjali*, is placed in a narrow-mouthed bottle and heated on a sand bath until reddish fumes appear. *Makaradhwaja* is deposited on the inner surface of the bottle on cooling. The bottle has to be broken and the *makaradhwaja* scraped off. The ayurvedic practitioners believe that *makaradhwaja* has special properties and is different from other preparations of sulphide of mercury or cinnabar; they assert that it is not an ordinary sulphide of mercury, but is in combination with gold. Neogi has shown that no gold is present in the preparation. Ghosh found mercury in the liver of a dog after it had been fed on *makaradhwaja* for several days. The authors conducted experiments with guinea-pigs with the object of discovering whether any absorption of mercury occurred after administration of *makaradhwaja*. In each of three instances they tied the pylorus, and in each of two others they placed a ligature at the ileo-caecal junction; the drug was administered through a window cut in the stomach. The contents of the animals' small and large intestines were examined separately *post mortem*, some twenty-four to thirty hours after the operation. In no instance could any trace of mercury be found in the large intestine. The authors presume that, if absorption had occurred, some mercury would have been excreted into the large intestine. In no instance were they able to find evidence of the storage of mercury in the liver. The authors draw attention to the fact that the mercury ion, highly diluted, has a stimulating effect on animal tissues; large doses of mercury cause loss of weight, a diminution in the hæmoglobin content and a decrease in the number of red corpuscles. Most preparations of mercury are rapidly absorbed; possibly in many cases too much mercury is absorbed. The authors observed considerable improvement in patients suffering from myocardial disease after the administration of *makaradhwaja*; they suggest that this drug may be rendered very slightly absorbable by the action of the gastric juice, so that minute quantities of mercury ions are taken into the system, with beneficial effects.

Treatment of Gonorrhœa.

S. LOMHOLT (*Münchener Medizinische Wochenschrift*, June 24, 1932) has investigated the effects of injecting various silver preparations into the male urethra as a means of quickly curing gonorrhœa. The most important point is the length of time such solutions should be retained. Neisser originally recommended twenty to thirty minutes, but as this was too painful it became the custom to advocate periods varying from five to fifteen minutes. Apart from the direct mechanical action of the fluids, the most important factor is the rate at which the solutions are converted into silver chloride by the mucous membrane. The author concludes from his work that 0·2% silver nitrate acts more quickly than a 1% protargol solution. The latter is preferable for recent cases in which pain is a prominent symptom, while silver nitrate is more valuable for chronic cases, because it tends to penetrate deeper in a shorter time. His results show that three daily injections, each lasting thirty seconds, are preferable to one single injection of five to ten minutes' duration. The course of the complaint was shortened and no complications were noted. In addition, the shorter time taken insures that many patients will carry out the treatment more thoroughly.

Basal Anæsthesia.

H. WEISBADER (*Münchener Medizinische Wochenschrift*, August 12, 1932) discusses the value of basal anæsthetics such as "Pernocton", "Sodium amytal" and "Nembutal". For minor gynæcological work no other drug is required, while ether in small amounts is added for major operations. The technique with "Pernocton" is as follows. A sedative is given the previous evening, morphine and atropine are given one hour before operation, and a quarter of an hour before operation "Pernocton", one cubic centimetre per 12·5 kilograms body weight. Ethyl chloride is given when the skin is incised, and if necessary ether is given by the open method during the operation. The author considers that "Sodium amytal" and "Nembutal" are similar in action, but that the dosage recommended by the makers is too high and is liable to be followed by veronal poisoning. The details of such a case are given. The after-effects of both drugs last for eight to ten hours, as compared with five to six hours with "Pernocton". This is not always to be desired, especially with elderly patients with deficient pulmonary ventilation. The main contraindications to the use of these drugs are elderly

patients with arteriosclerosis and high blood pressure, young children, and any with marked circulatory disturbances.

W. HEIM (*ibidem*) is enthusiastic regarding the value of "Pernocton" basal anæsthesia. While one cubic centimetre per 12·5 kilograms body weight is the usual dose, he has not exceeded four cubic centimetres as a maximum dose. The drug must be injected slowly and any untoward symptoms of collapse can be speedily combated with intravenous injections of five cubic centimetres of "Coramine". All the author's operations are now performed under "Pernocton"-ether anæsthesia. The results with goitre in particular have been very good. Unlike some observers, he has not found that as much as 80% of ether is saved, although the saving is considerable. In addition, "Pernocton" is cheaper than some other drugs. The great advantages of this basal anæsthesia are the lessening of pre-operative psychical shock, decrease in post-operative pain and the prevention of pulmonary complications following prolonged general anæsthesia.

NEUROLOGY AND PSYCHIATRY.

Treatment of Involutional Melancholia.

KARL M. BOWMAN AND LAURETTA BENDER (*American Journal of Psychiatry*, March, 1932) have investigated the treatment of involutional melancholia by ovarian hormone in the form of Squibb's "Amniotin", recording simultaneously the changes in blood pressure and blood chemistry. Two patients showed good recovery, two patients died of intercurrent disease, and three patients failed to show any response. In no instance was the blood chemistry appreciably altered; but the administration of ovarian hormone appeared to improve the patient's general condition and alleviate some of the distressing symptoms. Squibb's "Amniotin" is a water-soluble extract from the fœtal fluids of cattle, standardized in Allen-Doisy rat units, so that each cubic centimetre contains ten or twenty rat units. The substance was injected hypodermically in amounts of from twenty to forty units per day.

Masked Thyreotoxicosis Simulating Primary Neurosis.

SOLOMON GINSBURG (*The Journal of Nervous and Mental Disease*, October, 1932) reports upon the nervous and mental symptoms dependent upon masked thyreotoxicosis, and the response to treatment by radium and iodine therapy. He claims that judicious and efficient radium treatment has yielded better results than surgery and has been less productive of mortality. He believes that a condition of thyreotoxicosis frequently underlies neurotic states, cases of "nervous indigestion" and "neuro-circulatory asthenia". He claims that depressive psychotic states may arise as the result of the production of toxic thyreoid products, and that when such a state is accompanied by tachycardia, tremor, heat intolerance, excessive sweating, loss of weight, diarrhœa with or without thyreoid enlargement, the possibility of a thyreotoxicosis should never be forgotten. He has found in such cases, when the underlying cause is obscure, that clinical improvement following radium application over the thyreoid speaks in favour of an underlying thyreotoxic state.

Tremor of the Tongue.

MAX H. WEINBERG (*The Journal of Nervous and Mental Disease*, September, 1932) discusses the diagnosis of tumours in the region of the third ventricle. He believes that tremor of the tongue may be a sign of localizing value in cases presenting the syndrome of internal hydrocephalus, cerebellar asynergy and increased reflexes. The writer reviews the neurological signs in four cases of third ventricle tumours and endeavours to correlate them with the autopsy findings. All four patients presented a well marked lingual tremor. The writer thinks that the tremor was due to its close proximity to the basal ganglia, pressure being produced on the hypoglossal fibres in the genu of the internal capsule.

Streptococcal Meningitis.

KARL ROTHSCHILD (*The Journal of Nervous and Mental Disease*, October, 1932) reports a case of non-hæmolytic streptococcal meningitis with recovery, occurring in a male patient of twenty-nine. Examination of the cerebro-spinal fluid in this case showed a cell count of 12,500 and a globulin figure of +4; the culture yielded a low grade pleomorphic streptococcus of the non-hæmolytic type. The onset of the illness was acute. The coma lasted three weeks. The family history was without taint. Though the cause of the illness was never definitely determined, an infected tooth was regarded as the possible focus of infection. The patient was treated by intravenous injections of 500 cubic centimetres of a 5% glucose solution twice daily and by frequent spinal punctures. Recovery was complete.

Auditory Hallucinations in Children.

MAX LEWIN (*The American Journal of Psychiatry*, May, 1932) discusses the question of auditory hallucinations in non-psychotic children and their significance in the general problem of projection. The four children whose hallucinatory states formed the basis of this study, showed no acute or recognizable psychosis; although two of the boys were possessed of other symptoms which might later have become those of the schizophrenic reaction. All four boys were below average intelligence and two showed marked maladjustment to environment. Investigation revealed that these auditory hallucinations arose or participated in the fulfilment of the following functions: (*a*) defence, (*b*) enhancement of self-esteem, (*c*) satisfaction of instinctive cravings and of a desire for pleasure, (*d*) repression of unwelcome instinctive cravings, (*e*) self-punishment in expiation of a sense of guilt. Unlike the hallucinated insane, all four boys recognized that the "voices" originated in themselves. None suspected an external agency as the source of the hallucinations.

Spastic Pseudosclerosis.

CHARLES DAVISON (*Brain*, Volume LV, Part 2, 1932) presents two cases of spastic pseudosclerosis with full clinical notes and necropsy findings. The first patient was a male, aged thirty-two, and the second a female, aged fifty years. In both cases the illness ran a rapid and fatal course. It was characterized clinically by mental symptoms, pyramidal and extrapyramidal signs and muscular atrophies. The mental symptoms were those of amnesia, apathy and disorientation. The histopathological findings in the spinal cord were those of amyotrophic lateral sclerosis; but the extrapyramidal symptoms and the changes observed in the *globus pallidus* and related structures make the relationship of these cases to the more common condition of amyotrophic lateral sclerosis seem very doubtful. In addition the autopsy revealed atrophy of the cerebral convolutions, destruction of the ganglion cells in the third, fifth and sixth laminæ, and calcification of the vessels of the pallidum. The author gives a full discussion of these cases, with detailed references to similar cases in the literature. He differentiates his cases from multiple sclerosis, general paralysis, Alzheimer's disease and *dementia præcox*, and suggests the title of "disseminated encephalomylopathy" as being more clinically descriptive than the older term of spastic pseudosclerosis.

Lipiodol in the Diagnosis of Spinal Block.

H. H. MOLL (*The Journal of Neurology and Psychopathology*, July, 1932) reviews the use of lipiodol in the diagnosis of spinal block. He points out the dangers and gives full details for the technique of cisternal puncture. He goes fully into the radiological interpretation of results, his article being illustrated by numerous radiograms showing all grades of partial arrest and false arrest caused by the adherence of the lipiodol to the nerve roots. He notes that while manometric and serological examination of the cerebro-spinal fluid may serve to establish the correct diagnosis, there are many cases which might remain undiagnosed but for the unequivocal radiological evidence offered by lipiodol. He considers the introduction of lipiodol a perfectly safe procedure if properly carried out, but utters a warning in the case of inflammatory conditions of the cord and meninges. It is noted that partial or complete block may be brought about by other conditions than spinal tumour, namely, pachymeningitis, syringomyelia and *meningitis serosa circumscripta*. These conditions may always offer the greatest difficulty in differential diagnosis.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held on June 30, 1932, in the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. A. J. GIBSON, the President, in the chair.

Pituitary Tumours.

PROFESSOR H. R. DEW read a paper entitled: "Simulation of Pituitary Disease by Intracranial Lesions" (see page 771).

PROFESSOR C. G. LAMBIE, after briefly reviewing the history of the subject, said that various more or less well defined clinical syndromes had been ascribed to dysfunction of the pituitary gland. In the past it had been customary to speak of hyper-, hypo-, or dyspituitarism of the anterior or posterior lobe, and it had been assumed that each of these lobes furnished a single hormone, which might be secreted in deficient, excessive or normal amounts. In the light of recent work it was necessary to recognize the existence of several more or less independent activities of the pituitary gland, due in all probability to the action of several hormones. It was necessary to review the interpretation of clinical syndromes in the light of this work. Confining one's attention to the anterior lobe, there were at least three important activities to be considered. There was the growth-promoting factor whose action was direct. Next, the gonadotropic factors, which were probably two in number: (*a*) the factor responsible for the development of the gonads, sexual activity, ovulation, the development of the secondary sexual characteristics, and the sealing of the epiphyseal junctions; (*b*) the factor causing luteinization and the induction of those changes in the uterus which prepared it for the implantation of the ovum and the growth of the placentum. Failing pregnancy, these latter changes were followed by menstruation in the human beings. The action of these hormones was indirect, through the gonads.

The third factor was thyreotropic. It had been shown experimentally that the anterior lobe of the pituitary secreted a substance which, like thyroxin, caused the metamorphosis of neotonous amphibia, but, unlike thyroxin, its action was indirect, namely, through the thyreoid. Injection of pituitary extracts into mammals also caused hypertrophy of the thyreoid and microscopic changes indicative of hyperactivity. Either the same or another factor might be responsible for the control of fat deposition and the maintenance of normal basal metabolism. The obesity and lowered metabolism present in some pituitary disorders might be due to a deficiency of this hormone. In view of the marked similarity between the obesity resulting from pituitary disease and that which followed ablation of the gonads, it was possible that here again the action might be indirect through the gonads, although the possibility had also to be considered that ablation of the gonads might interfere with the elaboration of the pituitary metabolism factor, the action of which would then be direct.

Taking these three factors—growth-promoting, gonadotropic and thyreotropic—and examining the possible combinations resulting from their excess, deficiency or normality, it was found that there were twenty-seven different possible combinations. Each of these combinations might theoretically correspond to a more or less well defined syndrome, although such syndromes, many of which had been named after particular individuals who had described them, should be regarded as clinical abstractions, in view of the manner in which many of them merged into one another. Each individual case should be considered a distinct problem and interpreted in the light of our knowledge of the various activities of the pituitary gland. Nevertheless, it was of interest to observe that out of the twenty-seven different theoretical combinations it had been possible to find satisfactory examples of only eleven, and more or less doubtful examples of seven more, while for the remaining nine no clinical example had been found. Of the doubtful combinations, the existence of five was probable, although one of these types would be difficult to distinguish from a primary thyreoid and the remaining four from a primary gonadic affection. The two remaining doubtful cases were probably a very rare occurrence, if they existed at all.

An analysis of the unknown combinations showed that the following were incompatible or were at all events very rarely found together: sexual precocity with hypothyreoidism; hyperthyreoidism with dwarfism; hyperthyreoidism or hypergonadism *plus* hyperthyreoidism with normal or deficient growth. The absence of a triple excess syndrome was accounted for on grounds of probability alone.

DR. A. S. WALKER said that the neuro-glandular area in the region of the pituitary was of great interest in view of the fact that it was phylogenetically old. Here there was no question of functions of a higher type, such as were associated with the cerebral cortex. All the effects of lesions in this vicinity were upon metabolic and primitive mechanisms, with one exception, the affection of vision.

In considering pituitary lesions the physician interested in neurology was concerned chiefly with the reactions of these lesions upon, first of all, the gland structures, and secondly, the neighbouring nerve centres, and also with the problem of localization.

As regards the first of these, this has been described with great detail by Professor Lambie in his most interesting and comprehensive analysis of these endocrine disturbances, and little remained to be said. The changes noted might be due to an increase of hormonic influences or to a decrease of these or to a combination of both. An increased supply of pituitary hormones might cause positive growth anomalies, due to excitation of the anterior eosinophile cells, positive sex changes, such as virilism in females, due to excitation of the basophile cells, or glycosuria attributable in part at least to the posterior lobe. Decrease in hormones, as, for instance, caused by destructive changes, might cause diminution of growth activity, such as dwarfism and progeria, or of sexual activity, such as amenorrhœa, or the Fröhlich syndrome, obesity or *diabetes insipidus*. It must, of course, be recognized that these changes might be in part due to alterations in the function of the hypothalamic centres, and in this connexion the importance of the pituitary "portal system" referred to by Professor Lambie should be remembered. As regards these hypothalamic effects one was a little chary of touching on this involved and difficult subject. But a simple summary of the functions of the various cell groups in this part of the diencephalon was as follows. Of the chief cell groups lying in the floor of the third ventricle, two, the supraoptic and the *tuber cinereum* groups, in conjunction with the posterior pituitary gland mass, might be regarded as the anterior group or complex, parasympathetic in function. The posterior group, the supermammillary cells, were sympathetic in function. This concept gave two cell groups with opposed functions. Depression of the activities of one group might cause release phenomena due to the unrestricted influence of the other group. Thus depression of the anterior group would release the sympathetic group and cause the "sham rage" of experimental animals. Depression of the posterior group might cause such symptoms as hypothermia or drowsiness. These opposed symptoms were illustrated in the remarkable case just described by Professor Dew, in which somnolence succeeded attacks of loss of cortical inhibition as regards conduct. This sequence was obscure, but most interesting.

Depression of the activities of these nervous cellular groups might be caused by a definite lesion, such as a tumour acting by direct pressure or destruction, or by the cutting off of hormonic influences. The second and practical consideration was that of localization. This had really been dealt with most fully by Professor Dew in his illuminating address, but some summary of the points of interest to the neurologist might be given.

Firstly, there were the eye changes. Atrophy was the common finding, frequently unequal in the two eyes. The finding of bitemporal or homonymous hemianopia had been

described already. Most important was the perimetric examination of the fields of vision. This was one of the Cinderellas of clinical medicine. It should not merely be done in a perfunctory way; repeated careful tests should be made with objects of varying size and colour. Papillœdema was usually seen only in suprasellar tumours of the type of the cranio-pharyngeal cysts. Next there was X ray examination. Here the helpful findings were evidence of calcification, ballooning of the sella, or erosion of the clinoid processes. Hormone changes had already been referred to. They always occurred in pituitary tumours, sometimes in tumours of the neighbourhood, as shown by Professor Dew, but seldom in meningiomata. Age was very important to consider. A young patient, especially with growth disturbance, was the most likely subject of the cranio-pharyngeal cysts; meningioma occurred in middle age; and the not uncommon chromophobe adenoma was a disease of adult life. Headache was important. It was often bursting in character, with an intrasellar tumour causing pressure on the sellar diaphragm, but the existence of this type of headache did not imply that invasion beyond the diaphragm had not occurred, since there was frequently some coexisting optic atrophy, which meant disturbance of the chiasma. Glycosuria was not of much value in localization. It might be due to some other cerebral disturbance, or to too active a posterior lobe. It was found that in acromegaly glycosuria disappeared when this lobe was compressed. Obesity was not very helpful either, for it was not necessarily due to hypothalamic pressure. It might occur without the spread of a tumour above the sella, owing to the interruption of normal relations of the hypophysis to the nerve centres. Drowsiness was not of great use for accurate localization. As well as in cases of true pituitary tumour, it might occur with suprasellar growths or any others affecting this region by pressure. Parasympathetic and sympathetic disturbances might exist in bizarre combinations; their localizing value lay chiefly in their recognition as regional symptoms. Thus sham rage, or its clinical equivalent, meant invasion of the hypothalamus, so would also the opposite state of indifference, such as occurred in some cases of congenital cysts. Fits occurred only in pituitary tumours where extension of pressure or invasion of the diencephalon had occurred. Thus in one case, considered to be a suprasellar meningioma, attacks of fainting occurred, the patient's head drooped, his body became rigid, one arm was held out stiffly before him, and then there were a few clonic jerks. This suggested "fits" of the decerebrate variety. Finally, as shown earlier in the evening, distant lesions might, by sheer size and pressure, cause pituitary symptoms, and large pituitary tumours might invade the brain and cause a new group of confusing signs. These varieties of false localization should be borne in mind.

Finally, they came back near the starting point, to the importance of perimetry. Neuro-physician, neuro-surgeon and ophthalmologist should all take a vital interest in this procedure in any case of suspected lesion in the region of the pituitary. Amidst the confusion of the other varied signs of disturbed metabolism there stood out this definite and accurate landmark, on which all concerned should concentrate, the estimation of the fields of vision.

DR. H. R. G. POATE expressed his gratitude for the exposition of the subject of pituitary tumours given by Professor Lambie and Professor Dew. It was a subject in which he was particularly interested. During the third year of his medical course he had spent many hours at the abattoirs with an axe, splitting open ox heads in search of material. Dr. Poate had a patient suffering from acromegaly at present under his care. The patient had developed what appeared to be acute hyperthyreoidism. The thyreoid had become enlarged to a tremendous size and the basal metabolic rate had increased greatly. Tachycardia had developed and, eventually, fibrillation. Deep X ray therapy of the region of the pituitary had appeared to be the best mode of treatment. As a result the hyperthyreoidism had disappeared and the patient's basal metabolic rate had become normal. But he had been left with some damage to the heart muscle. The acromegalic features remained, but the progress appeared to have been held up.

Dr. Poate went on to speak of the meningiomata. He cited a case in which a man had gradually become blind in one eye. A diagnosis of tumour in the suprasellar region pressing on one optic nerve had been made. At operation a tumour the size of the top of the thumb, which was pressing on the nerve, had been removed. Within eighteen months there had been recovery of the sight of that eye. Such tumours were very difficult to deal with, and demanded the combined efforts of physician, surgeon and ophthalmologist. Together they must make the diagnosis and map out the correct course of treatment.

DR. OLIVER LATHAM spoke of chronic arachnoiditis sometimes simulating symptoms of tumours of the pituitary region. Only that week he had examined a brain which to the naked eye appeared affected with chronic basal arachnoiditis. The optic chiasma and tracts and surrounding regions were encased in thickened pia. Further examination showed that the pia had been invaded by a glioma (astrocytoma) of the left Ammon's horn, and which besides had involved almost the whole of the inferior temporal gyri and uncus. Professor Dew had laid stress on the importance of "anosmia", sometimes unilateral, as one of the earliest signs of tumours of the pituitary region.

DR. A. W. CAMPBELL said that the main lesson to be learnt from these two most excellent, interesting and instructive demonstrations was that the pituitary apparatus was much more complex than they imagined, and did not function as a gland alone. He was pleased that the representative of the neurologists, in the person of Dr. Walker, had put a diagram on the slate and that the nervous part of the mechanism had been emphasized, because the pituitary was essentially a neuro-epithelial apparatus. Dr. Campbell thought that they could better understand the multiplicity of symptoms if they thought of it as such. The nervous part of the mechanism might be regarded as a station that sent short distance messages to the pituitary body and probably long distance messages to other parts of the body. As Hughlings Jackson would say, the lesion might fall on any part of this system and it might operate in three different ways: stimulate, depress or destroy. According as it fell on the nervous mechanism or on the nerve fibre connexions, or on the gland, so would there be different syndromes. Awful as it would seem to suggest such a thing, there might be even more syndromes than Professor Lambie had mentioned. It had been suggested that the tendency to somnolence, obesity and alleged reduction of sexual power of the great Napoleon towards the end of his career had been due to dyspituitarism, and but for this disorder there might have been a very different result on the field of Waterloo.

DR. A. J. GIBSON expressed his appreciation of the two exceedingly interesting lectures. They were from three points of view most instructive. Professor Lambie's twenty-seven points provided much food for thought; it would take them a long time to get the gist of these points thoroughly digested. Dr. Gibson referred to a wireless talk by Professor Dakin concerning chromosomes; he had been bewildered to learn that their variations ran into millions. He was glad to know that the possible pituitary complexes ran only into twenty-seven.

Professor Lambie, in reply, thanked those who had taken part in the discussion. He said that there were undoubtedly many more syndromes than those he had mentioned; it was a depressing thought. Among matters that he had not discussed very fully were glycosuria and the galactagogue action of the pituitary. It was an interesting fact, in regard to glycosuria and increased flow of milk, that they were more commonly associated with acromegaly (when they did occur). Glycosuria could not be attributed to the posterior lobe; he had gone into this question some years ago and had concluded that physicians and physiologists had been misled by the experiments of Burn on the antagonism between the pituitary and insulin. The best explanation seemed to come from a consideration of the action of vasopressin on the capillaries. Tumours of the anterior lobe of the pituitary were those most commonly associated with glycosuria. According to Bouchard, 40% of cases of acromegaly had glycosuria at one stage. Since

coming to Sydney, Professor Lambie had encountered two cases of acromegaly with goitre, associated with marked exophthalmos and hyperthyreoidism, which might have been regarded as primarily of thyreoid origin if he had not been prepared to look for pituitary disorder. One patient had been operated on for goitre by Professor Dew and had later died of bronchopneumonia. At the subsequent *post mortem* examination a tumour of the pituitary had been found.

Professor Dew, in reply, thanked Dr. Campbell for his remarks and said that he (Dr. Campbell) had touched on a very important point. In all these tumours they were faced by the problem of whether the lesion was destructive or stimulating. There was much loose writing in regard to lesions of the nervous system. It was taken for granted by many that if the lesion was exerting pressure, it was therefore destroying. In this Hughlings Jackson's teaching on the dissolution of function was being neglected. Professor Dew expressed his thanks for the interest that had been taken in his paper.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Mater Misericordiæ Public Hospital, South Brisbane, on October 7, 1932. The meeting took the form of a series of demonstrations by the members of the honorary staff.

Dilatation of the Aorta.

DR. P. W. MACGREGOR showed a male patient, aged thirty-four years, who had complained three weeks previously of hoarseness of his voice. He stated that two years before he had had an anæsthetic for the removal of teeth and that his voice had been hoarse since then. He also complained of shortness of breath on exertion and pain in the chest, which was not particularly related to exertion. He had a cough, which was not paroxysmal and which was mainly present in the mornings. He had a very slight sputum which did not contain any blood. On physical examination the heart was found to be normal in size; the sounds were clear. Both lower lobes of the chest showed faint crepitations; the right side showed some dulness with doubtful bronchial breathing. On one occasion, after the patient had been riding a bicycle, a tracheal tug was very evident; when he was quiet this was not noticeable. Examination showed the urine to be clear; the sputum did not contain any tubercle bacilli, and the Wassermann test gave no reaction. On ordinary examination the throat appeared slightly reddened; the patient admitted being a heavy drinker and smoker. Dr. Foxton had examined the throat further and had reported that the right true vocal cord was immobile. The pupils were equal and reacted to light and accommodation. The systolic blood pressure in the left arm was 130 millimetres of mercury, in the right arm it was 140 millimetres, and the volume of the pulses at the two wrists was felt to be different. An X ray examination of the heart and large blood vessels revealed a dilatation of the aortic arch. The pulse rate was taken before effort, when it was 80 beats per minute; after exertion it rose to 120, and after ten minutes' rest it had only dropped to 110 beats per minute. The hoarseness was progressing.

The patient admitted having had a gonorrhœal infection fifteen years previously. He was married, with two children, and his wife had had no miscarriages. It was possible that he might have had a syphilitic infection as well. The prognosis was hopeless. Treatment took the form of large doses of iodide and advice to lead a quiet life. The question arose whether one was justified in making him leave his work.

Melanotic Sarcoma.

DR. B. L. W. CLARKE showed a female patient, who, four years previously, had had a black mole removed from the calf of the left leg. Dr. Duhig had examined the specimen and had reported that it was a melanotic sarcoma. Dr. Clarke had seen the patient. Fresh nodules had appeared. She had had twenty-four X ray treatments and eight radium treatments to each fresh nodule, and most of them had responded to radium. The patient had then gone to a quack for treatment, but in July, 1932, she had reappeared with a paresis of the right side of the body and with loss of the power of articulation. She had had further treatment, but was covered with nodules in every part of the body. It was four years since the first nodule was removed; this was a very unusual length of history for such a case.

Injury to Heel.

DR. J. A. LYNCH showed a boy who had had an injury to his heel six months previously. He was suffering from tenderness over a small area on the heel and pain on walking. Examination of the heel revealed no abnormality. An X ray examination showed that the epiphyses of the calcaneus were uniting normally. There were several transverse lines on the shaft of the tibia, which might be due to the deposit of lead or to syphilis. Blood examination revealed 4,980,000 red cells and 7,300 white cells per cubic millimetre, and a normal film. Platelets were normal. The Wassermann test gave no reaction. It was considered that the transverse lines might be due to a deposit of calcium during an epiphysitis. Dr. Lynch said that rickets frequently caused similar transverse lines, or they might occur as the result of severe illnesses and changes in the osteoblasts.

Hysterical Contraction of the Hand.

DR. G. W. MACARTNEY showed a woman who, twelve months previously, had sustained a Colles's fracture of the right wrist. She had been treated on a Carr's splint and the whole hand had been bandaged over the splint. When the splint was removed, the hand was in its present position. She complained that she had no sensation in the arm or the hand. The accident occurred while she was at her work, and she had received compensation. She had seen a number of medical men and had become hysterical at times. She now had a perfect example of the glove anæsthesia. There was fair movement of the thumb and she could hold a pencil. The question of a Volkman's contracture was discussed. The hand was not a *main en griffe*; it could be opened a little, but spasm occurred when it got a certain distance. It relaxed a little in sleep, but not completely. The anæsthesia was demonstrated and Carr's splint was mentioned only to be condemned in the treatment of a Colles's fracture.

Lesion of the Cerebellum.

DR. ELLIS MURPHY showed a female patient, aged forty-eight years, who had been admitted to hospital six weeks previously in a comatose condition. She was vomiting a fair amount and could be roused with effort. The head was buried in the pillow. Examination shortly after admission to hospital revealed very marked conjugate deviation of the eyes and head to the right. There was marked flaccidity of the limbs on the left side of the body, and definite spasticity of the limbs on the right; a double Babinski's sign was present. An attempt was made to do a lumbar puncture, but the patient was very fat and this was not successful. A cisternal puncture was done and clear fluid under slightly increased pressure was obtained. This fluid contained sugar. No globulin was present, there was no increase of cells and the Wassermann test gave no reaction. A diagnosis of apoplexy was made, with doubt as to whether the cause was a hæmorrhage or a thrombosis.

Dr. Murphy said that Collier stated that if a hæmorrhage had taken place, no treatment was of avail; if a thrombosis was present, stimulating treatment might do good. With this in mind, Dr. Murphy had given the patient digitalis, a little alcohol and large doses of thyreoid; she had improved considerably.

At the time of examination at the meeting the deviation to the right was disappearing. Nystagmus was still present and was more marked on looking to the left. There was still some weakness of the left rectus muscle. The paralysis of the left side of the face was improving. The

limbs on the left side of the body were still quite hypotonic, while on the right side it still required a definite amount of force to flex the limbs. The alteration in sensation on the right side was still present. The Babinski's sign had disappeared; the difficulty with swallowing had also disappeared.

The diagnosis appeared to lie between a tumour of the cerebello-pontine angle, with hæmorrhage occurring into it, or a primary vascular condition. The patient had given a history of headaches for some considerable time, but he had had no vomiting or giddiness. Dr. Murphy thought probably there had been a thrombosis of the posterior cerebellar artery which caused the condition.

Spastic Paraplegia.

Dr. Murphy also showed a man, aged forty-eight years, who, up till one year ago, had been quite well. His occupation then was planting bananas and he began to find that he could not get about the hills. From then on his legs had gradually become weaker and he had also noticed weakness of the fingers three months before. He was admitted to hospital and examination revealed a definite spastic paraplegia; there was a marked degree of paralysis of the legs; the knee jerks were exaggerated. The arms were normal and there was no sign of paresis or paralysis. The lower abdominal reflexes were absent. There were sensory changes below the nipples; there was no hyperæsthesia, but sensation to pain, heat and cold, and the vibratory sense were altered. The Wassermann test gave a strongly positive reaction and the patient was being given mercury and iodide of potash. A lumbar puncture was done; the fluid was under a tension of three hundred millimetres of water; there was no increase in protein, and only one cell per cubic centimetre; the Wassermann test applied to the cerebro-spinal fluid gave no reaction. A cisternal puncture was performed and lipiodol injected below the *foramen magnum*. The lipiodol went down to the lowest level, and an X ray examination of the spine revealed no abnormality. The fundi were clear, the pupils were equal and reacted to light and accommodation. The blood had since failed to react three times to the Wassermann test. The patient was still having iodide of potash and mercury, and was to have a course of "Bismol" or some similar preparation, and the patient was to be treated as if he were suffering from a meningo-vascular lesion. He appeared to be improving, but the condition had been going on so long that there were sure to be permanent changes in the tracts.

Perirenal Abscess.

DR. P. J. KELLY showed a boy who had had a boil on the left leg six weeks previously. Three weeks later he had complained of malaise; he had gone to bed and had been seen by a doctor. He was feverish and it was thought he might have typhoid fever; he was watched for about a week and the only thing found was slight tenderness over the left kidney. He was admitted to hospital with a temperature of 37·8° C. (100° F.). He did not complain of pain, but was tender over the left kidney. The temperature rose in the evenings and the boy had sweats. The tenderness became more severe and was working round to the front. No fullness or definite mass could be felt. The Widal test gave no reaction for *Bacillus typhosus* or for *Bacillus paratyphosus A* or *B*. A blood count revealed 15,000 leucocytes per cubic millimetre, with 86% neutrophile cells. One week later the leucocytes were 18,000 per cubic millimetre and neutrophile cells 92%. An X ray examination showed that the splenic flexure was displaced downwards, probably by a mass, perhaps a hypernephroma, and the stomach was pushed up. Dr. Stanley Roe had carried out a functional test on the kidneys and found that the dye appeared more sluggishly on the left side. The urine from the left kidney contained a very occasional epithelial cast, a few pus and red cells. Dr. Roe thought there was still a possibility of a cortical abscess of the left kidney, in spite of the almost clear urine, and had asked for an X ray examination to be made of the pelvis of the kidney.

Correspondence.

AN ECONOMIC COMMENTARY AND DIAGNOSIS.

SIR: On reading the scathing denunciation of the efforts of Dr. De Garis to enlist biological interest in the disorders of the body politic from the pen of a "Cobbler to his Last", one wonders if an attack of this sort should be accepted under the protection of a *nom-de-plume*. Whatever the merits or demerits of Dr. De Garis's thesis, there is no doubt that authoritative non-biological sociologists and economists state that a scientific foundation to their subjects cannot be established till biological investigations reveal the laws underlying social trends of behaviour.

The long-viewed sociologist sees a process of diverse human appetites and aspirations (studied by biology) obtaining satisfaction through the medium of human institutions (studied by sociology and economics). And compiling of the book of knowledge of life, where the root origins of booms and depressions and their economic and social complications are concerned, demands the pooling of specific facts from both these distinct sources of research.

If the reader of THE MEDICAL JOURNAL OF AUSTRALIA desires to know just where research to date stands in this connexion, he may gain an excellent foundation by reading the volume "World Chaos", by Professor William McDougall, the eminent biologist, sociologist and economist of Oxford and Harvard.

As a medical graduate who has devoted his life to academic sociology and economics, there is combined the type of investigator Dr. De Garis would desire more of our profession to emulate.

Among many striking assertions in the above-mentioned volume, *inter alia*, William McDougall tells us:

> I may add that a person who is in touch with many sources of information that are closed to the public, recently asserted in conversation that during a few days in the present summer (1931) our civilization tottered on the verge of collapse. I am not at liberty to give his name. I may only say that he is very widely and highly regarded for his prophetic and generally optimistic views of world affairs. It is only the biological and especially the social sciences founded on biology that can save us.

Continuing, he says:

> This sentence needs expansion. My thesis is that in order to restore the balance of civilization, in order to adjust our social, economic and political life to the violent changes which physical science has directly and indirectly produced, we need to have far more knowledge (systematically ordered or scientific knowledge) of human nature and of the life of society than we yet have . . . Briefly, then, we urgently need a well founded theory of evolution such as at present we lack; and we need knowledge of its detailed application to the human race. Secondly, we need the development of the social sciences, economics, politics, etc., for guidance in all social and political problems, in the face of which we stumble blindly along amidst a chaos of conflicting opinions. All of these need for their foundation some such knowledge of the construction of human nature and the principles of development. But we have no biology that can serve as the basis and the social sciences, are but a faulty sketch of a programme. In every branch is the same story. The economic book is full of iron laws. Yet in any true sense is there an economic law anywhere in sight?

Later McDougall writes:

> Arnold Tonynee-bee wrote: "Economic facts are obscured by human passions and interests."
>
> He should have written that "essential economic facts are human passions and interests" . . . It is well to remember when conditions are bad, men are without work, children short of food, that the malady from which society is suffering is plain ignorance (of how to manage affairs of economics by knowledge of laws of life).....

I would by every means divert our most powerful intellects from the physical sciences into research into the biological, the human and the social sciences, and our universities should be the main seats of such research. Especially would I at first concentrate the attack upon anthropology, the science of man concerned, in the broadest way. It would take 20 years to train the personnel and get them working on their problems. Then, as they began to bring in their results, part of the available intellectual energy would be concentrated in the effort to build the social sciences, especially a *science of economics*, on the basis provided by the anthropological research. Can we afford to wait so long? Can our civilization survive in the meantime? I don't feel sure! but I hope it may, for I see no alternative measures that offer hope for its salvation.

In reviewing the motives underlying the letters of Dr. De Garis and "Cobbler", who is more comfortable in the horizon of his last, there is no doubt Dr. De Garis is not alone in her search for biological meaning of the many futilities in an otherwise glorious civilization. Her attack on this problem rather as an amateur economist than a biologist, however, robs her argument and example of half its force, as "Cobbler" has sensed. In entering this discussion it is the desire of the writer, apart from considerations advanced in this letter, to simply argue that monetary problems stressed by Dr. De Garis as primary are in fact secondary, and symptoms rather than signs to deeper economic trends.

How recourse to facts of science not envisaged by the industrialist or economist who has not lived among them, can reveal the truth of this assertion, it is proposed to discuss in a later communication, if space can be provided for the purpose.

Yours, etc.,

FRANK TRINCA.

96, Balaclava Road,
Caulfield,
Victoria, S.E.7.
November, 1932.

SIR: It is with a feeling of considerable satisfaction that I read the letter of Dr. Mary De Garis in your issue of the fifth inst. The satisfaction was not the result of the matter contained in Dr. De Garis' letter, but arose from the fact that you had the courage and wisdom to open your columns to a discussion on economics.

It must surely be apparent to all who have been in medical practice for some years that a great deal of disease and suffering has at least some roots in the economic conditions of the people.

Life to most men and women of middle age is a continual and hard struggle with a constant fear of the future—this fear alone is enough to bring about innumerable disorders of mind and body.

It is, therefore, incumbent upon all medical men to study economics, for such study will aid in the development of the proper function of the medical science of the future, that is, prevention as well as help in solving present difficulties.

I must say that I agree with many of Dr. De Garis's remarks, but when she attempts to diagnose our economic ills as being due to faults in our monetary system, she displays a sad lack of economic knowledge.

Her letter is evidence that it is no easier for a doctor to prescribe for the ills of the political body than for an economist to prescribe for the ills of the human body. Her commentary clarifies nothing and her diagnosis is out of harmony with facts. Society does not ignore or neglect the effects of time. Dr. De Garis herself draws attention to the fact that "the wise man allows for depreciation and the financier claims compound interest". Surely in each case the effect of time is taken into account. Most people would agree that our money system is imperfect. The difficulty is encountered when one attempts to devise a money which would be perfect, and I am utterly at a loss to visualize one "which allows for the effects of time, which recognizes the wastage, as well as the increase, by time". Money she defines as "mainly book entries", "mainly overdrafts," and "debts". Now the man or woman who could make a book entry, an overdraft, or even a debt "allow for the effect of time, recognize wastage, as well as the increase, by time" would indeed be superhuman. We had better not wait the advent of such a being before tackling the problem.

Your correspondent complains that "the pound is anything but stable". It cannot and should not be otherwise. Two of the functions of money are to regulate supply and prevent waste. Neither of these virtues could inhere in a medium which always exchanged for a fixed quantity of a given article. Suppose that crinolines had not varied in price since the time when it was profitable to produce them, we would have crinolines produced in quantity today when no one wants them.

With a flexible currency an inventor, by increasing supplies, has to accept a lower price for his products and so shares his advantage with all who buy his goods. He increases the incomes of all his customers because the currency measure varies with a variation in demand or supply. When supplies fail, prices rise. In that way some of the loss of the producer is made up; the public are helping to carry his burden and labour is enlisted to increase the additional supply requirements in the future. Surely this is a wonderful and beneficent arrangement, arising out of the fact that money is not rigid, but more or less adapts itself automatically to changing conditions.

Your correspondent says that "until primary industry pays unemployment must persist, and primary industry cannot pay until debt is wiped out. Debt can only be wiped out by providing a credit money". There is no reasoning here; only assertion.

Primary industry pays now. Almost any industry pays better than idleness. The trouble is largely due to the Government having handed over the profits of primary to those engaged in the secondary industries. To state that it does not pay to produce a certain class of goods is merely to say that such goods cannot be exchanged for a satisfactory quantity of other goods.

Surely no one can be surprised at our farmers being dissatisfied when we reflect how the goods our farmers wish to purchase have been made artificially dear by Customs taxes and embargoes. The Government cannot protect the farmer; therefore it ought not to protect anyone. All ought to be treated alike. If that were done, a big step would have been taken towards solving the difficulties of the farmer and the solution of the unemployment problem.

Let us undo the obvious injustices which exist in our social order before we introduce some undefined, vague and fantastic new currency, woven out of the stuff that dreams are made of.

Yours, etc.,

PAUL G. DANE.

110, Collins Street,
Melbourne,
November 29, 1932.

THE FUTURE OF MEDICINE AS A PROFESSION.

SIR: At a time such as the present one must sympathize with "One Too Many", and also thank him for his letter, and I would like to draw the attention of the Australian members of the British Medical Association to the report of the committee appointed by the British Medical Association, England, to investigate the abuse by patients who are able to pay private fees, of the benefits of the public hospitals. Also, I would like to quote two paragraphs from that report of their findings (*vide The British Medical Journal* Supplement, March 5, 1932—Report by the Joint Committee of the British Hospitals Association and the British Medical Association):

> ii. Institutional treatment has largely displaced treatment in the patients' own homes, and this tendency is likely in the future to increase. Mainly as a consequence of the foregoing, social status and private means are coming to be regarded less and less

by members of the public as reasons why any patient should or should not go to hospital.

iii. The substitution of institutional for home treatment has narrowed the field of private medical practice in the case of consultants and specialists. In consequence, these practitioners find it increasingly difficult to earn their living, and in many instances this is reflected in an increase in their fees to private patients.

In answer to those gentlemen who advocate the adoption of intermediate hospitals in conjunction with the public hospitals I would say that we should make the present public hospitals less attractive. Unless some alterations be made we will still be unable to fill our intermediate beds and be unable to deter people who are capable of paying small fees from entering the public portions of the hospitals. To remedy this disease of the public mind I would suggest several reforms.

1. To revert to the brown or blue blankets.

2. The red coverlet instead of the white embroidered counterpanes.

3. Stretcher beds, but not those of a type so low as to make nursing difficult and arduous.

4. Heavy porcelain and enamel plates, cups and saucers.

5. The wards to be made less attractive.

6. Hospital uniforms to be strictly observed. No silk underclothing, nightdresses or dressing gowns. The patients should not be allowed to wear their own clothes, but must be kept rigidly to the hospital uniforms without exception, wool and cotton garments.

7. Meals to be of a nourishing quality, but more frugally served—in fact the wards and meals should closely follow the workhouse style.

8. Maintain the high efficiency of surgical and medical treatment of the patients by the use of the latest surgical equipment and proved drugs whose therapeutic values are known.

9. Special branches to be well equipped.

In my opinion at present we give the sick poor something they do not seek, and at the same time encourage a class of patient who should not occupy these beds. It is on account of the present luxurious state of our hospitals that has made them over-capitalized and consequently they are unable to find the money for their upkeep. How embarrassing it is to a poor patient to be placed next to a patient of means, who, though unwilling to pay for private attention, is ready to make a display in the ward by wearing silks *et cetera* and is always surrounded by expensive flowers! On visiting days this patient is seen to be visited by many rich relations and friends, also to the embarrassment of the friends at the next bed. The sick poor do not need our present luxurious accommodation as provided in our general hospitals, nor do they demand it.

Finally, I would say that we can still treat the sick poor in the same efficient manner and make the hospitals less attractive to those who can pay, and, moreover, pave the way for the intermediate hospital. By these means, too, we would educate the public to pay for what they receive, and the profession would not be called upon to do so much work as it does for nothing.

Yours, etc.,

JAS. J. WOODBURN, M.B.

185, Macquarie Street,
Sydney,
December 7, 1932.

THE NEW SOUTH WALES MEDICAL BOARD.

SIR: The late members of the New South Wales Medical Board are forwarding to the President of the New South Wales Branch of the British Medical Association a communication, of which the enclosed is a copy. I have been requested by the members in question to ask you to be good enough to print the communication *in extenso*.

Yours, etc.,

G. H. ABBOTT,
(Late Secretary to the New South Wales Medical Board.)

Sydney,
December 17, 1932.

December 17th, 1932

The President,
The British Medical Association,
N.S.W. Branch.

Sir,

The late members of the N.S.W. Medical Board feel that a brief statement of their case should be placed before the members of the N.S.W. Branch inasmuch as some misconception of the real facts is believed to exist.

On November 29th, the Minister of Public Health despatched the following communication to each member of the Board individually:

Dear Dr. . . . . .

In connection with my administration of matters pertaining to public health, it has been necessary for me to review past activities and arrangements that have been in operation for some considerable time, and as a result the belief has been forced upon me that in very many directions there is need for an entire re-arrangement of existing conditions.

In the course of my enquiries I have given some consideration to the functions of the New South Wales Medical Board, created under the authority of the *Medical Practitioners Act*, 1912. While deeply appreciative of the work that this honorary body has undertaken during the period they have been Members of the Board in question, I consider that the time has now arrived for a general reconstruction of the Board, and to that end I am firmly of the opinion that in the public interest, the Board, as at present constituted, should cease to function.

To permit of reconstitution and the appointment of a Board comprising not more than five Members, I deem it expedient for the appointment of all Members of the existing Board to be terminated. The position would be considerably simplified if the Members of the Board tendered their resignations, and I shall be glad, therefore, if you would kindly give consideration to this suggestion, and favour me with early advice of your determination.

Yours faithfully,

(*Signed*) R. W. D. WEAVER,
Minister for Public Works and Health.

This was the first and only communication which passed from the Minister to the Board.

On receipt of the above communication the available members of the Board met, and noting that no reason had been given for asking for their resignations, decided to discuss the matter at a full meeting of the Board and requested the Secretary to forward the following letter (dated 5th December) to the Minister:

Dear Sir,

The available Members of the New South Wales Medical Board have requested me as Secretary of the Board to acknowledge, on their behalf, the receipt of the identical letters which you have addressed to the Members of that body, suggesting their resignation from the Board.

The Members are impressed with the importance of the communication and do not desire to deal with it until they have had an opportunity of discussing its terms with one another. At the present time all the Members of the Board are not available but an opportunity will occur for the consideration

of the communication at the next monthly Meeting on December 14th, after which the Members intend to furnish you with a reply.

Yours faithfully,

(*Signed*) G. H. ABBOTT,
Secretary, New South Wales Medical Board.

On December 10th, a statement appeared in a daily paper purporting to emanate from the Minister and indicating that "about seven members of the Medical Board would have to retire under the policy adopted by him for the reason that they had reached or were over 65 years of age." This was the only information that reached the Members of the Board that the question of age had been considered by the Minister.

As indicated above, the Members of the Board had informed the Minister that they would consider the matter at the monthly meeting on December 14th, and would then reply to his communication. But in the morning Press of December 14th, it was announced that "Mr. Weaver had issued a minute which had been signed by the Governor declaring vacant all seats on the Board, and had nominated seven doctors to form the reconstituted Board." On making inquiries at the proper quarter, we verified this statement.

Yours faithfully,

(*Signed*)
EDWD. J. JENKINS.
W. H. CRAGO.
G. H. ABBOTT.
J. A. DICK.
W. G. ARMSTRONG.
A. MURRAY-WILL.

MODERN OPTOMETRY.

SIR: Your leading article in the last issue of the journal will be read by the profession with great interest, and particularly so by ophthalmic surgeons. It affords me great pleasure that you have touched on a subject which hitherto has received little ventilation.

There is no doubt that the general practitioner has in many cases failed to cooperate with the ophthalmic surgeon to the extent which is necessary for the public welfare. The specialist, I feel, will always meet the suggestions of the general practitioner in regard to fees.

You make a statement which will not be generally admitted by ophthalmic surgeons, that even some optometrists can accurately estimate refraction. Your reference to the casual way in which some busy ophthalmic surgeons have treated patients does not necessarily mean that diagnosis or treatment has been faulty, for it is a general principle that the more skilled the operator in any field, the greater speed with which his work is performed.

The thanks of the whole profession are due to you for the attitude which you have taken in regard to these matters.

Yours, etc.,

D'ARCY WILLIAMS.

193, Macquarie Street, Sydney,
December 13, 1932.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", page xvi.

BRISBANE MATER CHILDREN'S HOSPITAL, BRISBANE, QUEENSLAND: House Physician, House Surgeon.

HOMŒOPATHIC HOSPITAL, ST. KILDA, MELBOURNE, VICTORIA: Senior Resident Medical Officer.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer (male).

THE PUBLIC SERVICE BOARD, SYDNEY, NEW SOUTH WALES: Assistant Medical Officer of Health (male).

THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Resident Medical Officer.

WESTERN SUBURBS HOSPITAL, SYDNEY, NEW SOUTH WALES: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chilliagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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